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THE AMERICAN PHYSICIAN IN 1846 AND IN 1946

A Study in Professional Contrasts

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Centenary celebrations afford a natural occasion for comparisons of the old and the new. The century which has elapsed since the Northern Medical Association of Philadelphia was founded in 1846 was one in which most aspects of civilization changed with unprecedented rapidity, and medicine was no exception to this rule. Hence the contrast between the status and activities of the American physician in 1846 and in 1946 involves much that may seem unusual to the modern observer.

The glance backward may also reveal some things which are of more than antiquarian interest, for certain problems persist even through a century of change. The most commonly aired grievance of American physicians during the 1840's, for example, was the difficulty of collecting fees. "It runs me almost crazy," wrote a successful practitioner about 1840, "to think that with hundreds upon hundreds due me professionally I find the greatest difficulty in raising a simple fifty dollars." [1] No doubt there has been improvement in this respect; but rumor has it that even now there is occasionally some difficulty of this sort, and the whole matter of professional income is involved in current discussions of health insurance. The earlier experience of members of the medical profession with this or with any other persistent problem invites comparisons which may throw light on contemporary situations.

Many who contemplate the past—and who does not do this occasionally?—go to one of two extremes. Some accept uncritically the modern faith in progress and assume that everything was pretty backward until they came along, while others reverse this attitude and succumb to a nostalgia for the good old days. In 1925 a Maine physician wrote this inquiry: "I wonder if the medical man is held in as high esteem, or is as important a factor in the community, as was his confrère a half century ago?" The reply was a negative one: the questioner was convinced that the doctor of 1875 had much the better position.[2]

For a real answer to his inquiry, of course, one must examine the evidence. A wealth of material immediately appears, pointing in the opposite direction. Whether one sticks to the seventies or goes back to the forties makes little difference, for a definite improvement in the status of members of the American medical profession did not set in until almost 1900. There were no public opinion polls then, by which to measure attitudes toward a profession; but the difficult position of physicians may be easily illustrated by random references. The newspapers of the forties and fifties, for example, sometimes ridiculed medical students and medical practice. Occasionally they went to extremes of condemnation. Thus the Philadelphia *Item* denounced (1858) the "poisoning and surgical butchery" which were common in practice,[3] while in the next year a large daily paper declared that the whole medical guild was "a stupendous humbug." [4] Many lay critics had as low an opinion of professional behavior as they had of professional skill. "It has become fashionable," declared the editor of the *Cincinnati Medical Observer* during the fifties, "to speak of the Medical Profession as a body of jealous, quarrelsome men, whose chief delight is in the annoyance and ridicule of each other." [5] Alarmed by such attitudes, physicians had begun in the thirties to deliver addresses on "The Present Position of the Medical Profession." Two decades later they employed such frankly discouraged headings as: "To What Cause Are We to Attribute the Diminished Respectability of the Medical Profession in the Estimation of the American Public?" [6]

It is clear, after even such casual glances at the record, that all was not well with the profession a century ago. Granting that there still may be some cynicism about medicine, it is hard to imagine a daily paper referring to current surgical intervention as mere "butchery," or condemning professional practice as "a stupendous humbug." Compare with these epithets, for example, the respectful manner in which both contemporary newspapers and movies usually deal with physicians. About a decade ago there was a series of films which portrayed medical personnel in almost glamorous colors. These were embellished with such titles as "Men in White," "Women in White," "The White Parade" and so on. For a while anything "in white" was good for boxoffice returns. There were also such laudatory pictures as "Pasteur" and "The Magic Bullet," to say nothing of the late lamented "Dr. Kildare." If Hollywood is to be trusted, the medical profession has today a far higher position in public esteem than it did in great-grandparents' days.

Read at the Centennial Celebration of the Northern Medical Association of Philadelphia, Dec. 5, 1946.

From the University of Pennsylvania School of Medicine and American Council of Learned Societies.

1. Shryock, R. H.: Letters of Richard D. Arnold, First Secretary of the American Medical Association, Trinity College Historical Society Papers 18-19, Durham, N. C., Duke University Press, 1929, p. 21.
2. Mann, F. W.: The Public Status of Our Profession, J. Maine M. A. **16**: 137, 1925.
3. Philadelphia City Item, Nov. 6, 1858.
4. M. & S. Reporter **1**: 356, 1859.
5. Editorial, Cincinnati M. Observer **2**: 129, 1857.
6. Editorial, M. & S. Reporter **1**: 141, 1859. There were many other such titles; Bauer, L.: On the Declining Relations of the Medical Profession to the Public, Cincinnati M. Obs. **2**: 97, 1857.

To what circumstances may one attribute the uncertain status of physicians a century ago? There is no one simple answer to this, any more than there is a single explanation of professional prestige at the present time. Various factors must be noted, if one is to understand the position in which physicians found themselves at the time this association was founded.

In an analysis of the status of any profession, one first thinks of the original process by which personnel is selected. One envisages today the years of college education and the standards of grade A medical schools, all of which tend to prevent the lazy or incompetent student from taking the Doctor of Medicine degree. But in 1846 almost any man with an elementary education could take a course of lectures for one or two winters, pass an examination and thereby automatically achieve the right to practice medicine by state law. Since the old training under an experienced physician (preceptor) had been gradually abandoned, doctors could be turned out in less than a year. As late as 1900 there were medical schools which admitted students who could not gain entrance to a good liberal arts college. To be sure, some college graduates went to the better medical schools; but a study made at Yale in 1850 indicated that most graduates preferred law and theology, and that those who did enter the field of medicine were usually not of high standing in their classes.[7]

It would be of interest to inquire, in passing, whether the first women to secure medical education were of superior caliber. One might expect this, in view of the opposition they had to overcome. Most physicians and laymen were shocked at the very thought of a "doctress"; but after the Woman's Medical College was opened in Philadelphia in 1850 some newspapers encouraged the idea, and there was occasional support even in the medical journals.[8]

Whatever standards were maintained by the few "female" colleges, most schools admitted youths without higher education and with uncertain secondary school backgrounds. In 1854 a candidate requested admission to the Savannah Medical College on the impressive ground that he was proficient in the use of his own language. A professor in reply said that it would help if the student also knew something of the classics but added hastily that he could pick this up "with perfect ease" while attending the lectures. The student was invited to enter and reminded that he could not find a better college in that part of the country.[9]

This last remark was significant. The Savannah College was apparently a proprietary school, even though a superior one, and the practitioners who owned it needed the tuition fees. By 1850 the original university schools were pressed by the competition of proprietary institutions that sprang up all over the country. This proliferation has been ascribed to the increase of population and the consequent demand for more practitioners than the old schools could supply. But since the country already seemed oversupplied with physicians by the fifties other influences must have been at work. The late Dr. William H. Welch believed that the English medical schools (without university connections) set a bad example here. But the London schools were at least associated with hospitals, while American institutions sometimes lacked this advantage. The truth is that there was money in a successful medical college, since the owners maintained it simultaneously with their private practice. The lower the standards, outside a few main centers, the higher were the registration figures and the profits. No wonder that schools multiplied, and that intense jealousies developed between the members of rival medical faculties.

Competition from mediocre schools hurt even the better institutions. When Pennsylvania tried to raise its entrance and course requirements during the forties, many youths were reported to be leaving for "easier quarters." The editor of the *Item*, already quoted, declared that even the Philadelphia students were coarse, crude and ignorant; a New York paper added the adjective "licentious" in describing the young medicos of that city.

Due allowance may be made for editorial exaggeration in such statements. But since they were supplemented by ample testimony from leading medical practitioners themselves, it is clear that the recruitment of the members of the profession was a rather casual process. This was of grave concern to professional leaders, and their efforts to form state medical societies were motivated in part by the effort to reform medical education. This was desired not only to provide better training per se but also to overcome the jealousies of rival schools. The same motives inspired the founding of the American Medical Association in 1847.[10] The national body encountered great difficulties, however, in seeking reform. Influential practitioners throughout the country were interested in proprietary schools and opposed standards which would have eliminated them. When Oliver Wendell Holmes, for example, condemned what he termed "inferior schools, wrongly located," he raised a storm throughout the South and West. Medical professors in those areas viewed this as a sectional attack on themselves and ridiculed Holmes's "highfalutin Boston notions."

Tied in with low standards in the schools was the general laxity of state licensing laws. Early in the nineteenth century certain states had enacted fairly good statutes, which provided for examination by state medical societies or boards. But it proved simpler to license automatically all who graduated from the schools; and as the latter deteriorated, licensing became almost a farce. Some reformers advocated federal licensure,[11] while others considered that the M.D. degree had become meaningless and urged that it be replaced for able men by some such designation as M.A.M.A. (Member, A. M. A.).[12] This would not control licensing, but would at least indicate the real physicians. Such protests achieved little until almost the end of the century, when the impact of scientific progress enabled the national Association to initiate a really effective reform movement.[13]

Another problem which faced medical societies a century ago was the relation of one practitioner to another. After the student had taken his degree, such as it was, he found himself in sharp competition on entering practice. By 1860 there was 1 practitioner for every 572 persons in the population as a whole.

7. Hooker, W.: Report of the Committee on Medical Education, Tr. A. M. A. **4**: 409, 1851.
8. Shattuck, G. C.: The Medical Profession and Society, Boston, D. Clapp & Son, 1866, p. 29.
9. Shryock,[1] p. 68.
10. Fishbein, M.: History of the American Medical Association, J. A. M. A. **132**: 637 (Nov. 16) 1946.
11. New York J. Med. **5**: 415, 1845.
12. Sponsored by the New Jersey State Medical Society in 1858; M. & S. Reporter **1**: 283, 1859.
13. The detailed story of medical education before 1860 is given in Norwood, W. F.: Medical Education in the United States Before the Civil War, Philadelphia, University of Pennsylvania Press, 1944.

The ratio was apparently higher in rural than in urban areas—just the reverse of the present situation. The rural physician was then in great demand and suffered few such disadvantages as he does at present. In the North (relatively urban) there was 1 physician to 534 inhabitants; in the South, 1 to 526, and in frontier states, 1 to every 417. The general average of 1 to 572 may be compared with that of 1938, which was 1 to 764.[14] The excess of physicians was such that many young graduates failed to secure a decent practice and abandoned the profession. The pressure of competition not only caused bitterness against sectarians but also created friction within the regular profession.

Prior to about 1800 there had been no rules to the game, and physicians even condemned one another in the newspapers. A famous illustration of professional friction in Philadelphia had been Benjamin Rush's newspaper debate with colleagues over the best methods of treating yellow fever. Laymen took sides, and in the course of this debate one critic referred kindly to Rush's method as "one of those great discoveries which are made from time to time for the depopulation of the earth."[15] Rush denounced his colleagues, resigned from the College of Physicians and viewed himself as a much-abused figure. This was typical of many professional quarrels.

These difficulties were not to be entirely explained by the competitive factor. This affected lawyers and business men as well, yet physicians were notorious for the peculiar rancor of their quarrels. Perhaps the vital nature of medical work made for unusual bitterness between rivals. Many a doctor, at a time when medicine was much more of an art than a science, honestly believed that competitors were killing their patients rather than curing them. Or, if the rival was not actually liquidating his clientele, he was doubtless doing them no good and overcharging them in the process. The welfare of the public as well as self interest demanded that he be exposed.

It was gradually realized, however, that the scandal of medical quarrels was injuring the reputation of the profession. Even Rush, a born controversialist, sensed this in his last years. He then pleaded that physicians should cease to condemn one another just because of different theories of practice and urged that "the whole odium of the hostility of physicians" be ascribed to "their competition for business and money."[16]

Medical societies, fully aware of the frictions resulting from competition, sought to standardized principles of professional behavior. Percival's "medical ethics," adopted in London toward the end of the eighteenth century, was the basis of the code accepted by the American Medical Association at the time of its founding, and this English statement was also largely incorporated in the principles accepted by state and local societies.[17] These codes expressed certain general ideals and stated the duties of physicians to patients, to each other and to the public. They have since been criticized as providing "trade union rules" or "books of etiquette" rather than true ethics. Some critics object to the limitations set on criticisms of one physician by another, lest these shield the mediocre man at the expense of his patients. The codes may need overhauling today to fit them to modern conditions,[18] but any one who recalls the time when physicians did boast openly of their own merits and of the inferiority of their rivals will recognize the real service which these statements provided.

Even after medical societies had been organized there was no little recrimination within the profession. In 1851, for example, the therapeutic claims of a certain physician were attacked on the floor of a state society. In retaliation the man then published a pamphlet referring to the "meanness and baseness" of the society's procedure, adding that the state organization contained only "the pest boys and inexcusable ignoramuses" of the profession. A member of the society, writing to a colleague, began by inquiring: "Have you seen the dirty pamphlet of Dr. X? . . . The more I reflect on this man's attack on the state society the more angry I feel. . . . After I read his account of pneumonia and his never having lost a single case out of 179! I might have swallowed his 'obstetricy' (where did he get that word?) but I choked at his pneumonia statistics. . . . I wish no controversy with such a man . . . but I shrink from no responsibility."[19] One can see from such correspondence that the officers of early medical societies had their hands full in preserving professional harmony.

Long before ethical codes were adopted the early medical societies had sought to deal with the strictly economic factor in competition. They realized, apparently earlier than did business interests, that uncontrolled competition in an entirely free market was not in the interests of competitors. Perhaps the guild tradition in medicine had never permitted the acceptance of a *laissez faire* philosophy so extreme as that embraced by merchants. At any rate, a prime motive in the founding of the pioneer state medical society (New Jersey, 1766) had been the standardization of fees. The fee bill issued by that society listed charges of 25 cents and up for calls in country practice, according to mileage, and such other items as $3.50 for deliveries. When it is recalled that annual tuition at Princeton was then only $4, these charges seem by no means low.[20]

Much the same comment may be made on the fee bills published by medical societies about seventy-five years later; that is, for the era just before the Civil War. Between 1835 and 1860, a fee of 50 cents or $1 was common for an office call at the physician's home in town practice, and rates were higher in the large cities.[21] These charges were at least the equivalent in purchasing power of $2 to $5 at the present time. Such comparisons are difficult to make for home

14. Eighth Census (Washington, D. C., 1860), pp. 670 and 677; cited by Stern, B. J.: American Medical Practice: In the Perspectives of a Century, New York, Commonwealth Fund, 1945, p. 63. The ratio of physicians to population remains higher today in the United States than in Europe, but contrasts are not so extreme as a century ago. An American Medical Association committee reported in 1848 that this ratio was five times as great here as in France (Fishbein, M.: History of American Medical Association, J. A. M. A. **132**: 852 (Dec. 7) 1946.

15. Cobbett, W.: The Rush-Light, New York, Feb. 28, 1800.

16. Gross, S.: Lives of Eminent American Physicians, Philadelphia, Lindsay & Blakeston, 1861, p. 314; cited by Butterfield, L. H.: Benjamin Rush: A Physician as Seen in His Letters, Bull. Hist. Med. **20**: 138 (July) 1946.

17. Leake, C. D.: Percival's Medical Ethics, Baltimore, Williams & Wilkins Company, 1927, p. 33.

18. That of the American Medical Association has been revised several times and is now being reconsidered again by the Judicial Council; Fishbein, M.: History of American Medical Association, J. A. M. A. **132**: 783 (Nov. 30) 1946.

19. Shryock, R. H.: Selections from the Letters of Richard D. Arnold: Medical Series, 1834-1875, Bull. Johns Hopkins Hosp. **42**: 156, 1928, p. 174.

20. Marsh, E. J.: An Outline History of the Medical Society of New Jersey to 1903, Proc. New Jersey Hist. Soc. **60**: 5 (Jan.) 1942.

21. Rosen, G.: Fees and Fee Bills: Some Economic Aspects of Medical Practice in Nineteenth Century America, Bull. Hist. Med., 1946, supp. 6, p. 11.

calls, because the physician then charged in proportion to the distance traveled. The rural doctor also charged for medicines, so that it is hard to isolate the costs of professional services from those for drugs. Thus Dr. Tomlinson Fort of Milledgeville, Ga., included such items as the following in bills for 1841:[22]

Visit, 4 miles in rain, and med.	$ 7.00
Visit, nocturnal, 4 miles	8.00
Visit, 4 miles, $5.00 med., for gonorrhea	15.00

If one at least doubles these figures in order to estimate their purchasing power today, the fees seem relatively high.

Due allowance must be made here for certain variables. Dr. Fort's bills went to a planter who was probably wealthy and was perhaps charged accordingly. Bills sent to the rank and file may have been lower. Actual charges to most patients, moreover, were sometimes less than those stated in the fee bills. There were complaints to this effect, for a published schedule was after all only a gentleman's agreement. But, keeping in mind all these factors, it seems likely that fees in general practice a century ago were little if any lower in real value than are current charges.

This is surprising when one recalls, first, how low the costs of medical education and equipment then were; and second, the common impression that rising professional costs have forced fees up since that time. Present protests against medical costs are not caused by higher fees in general practice but result rather from the many specialized services now required.

Had the average total cost of medical care been as high in 1846 as it is at present, it would have been quite impossible for the great majority of patients to have met it. The average, per capita real income then was less than half what it is today.[23] In other words, the ability to maintain present levels in medical costs—even as well as is now done—results from improvements in living standards all along the line.

Total medical costs were relatively low in 1846 because most persons were served simply by the family physician. Those in and near towns could reach pharmacists and dentists, and they occasionally resorted to consultants; but most rural folk, who then made up about 85 per cent of the population, received only bills for office or home calls from a single physician. Many of these charges, moreover, were not paid—a simple way of keeping down medical costs.

Difficulties in collecting fees inclined some physicians to accept so-called "contract practice." A physician would care for a family in return for a fixed payment in advance, perhaps for as little as $5 or $10 a year. There were then few opportunities for salaried positions, and contract work may have appealed to practitioners in need of income. Similar procedures were sometimes followed in Europe, where they had had a long history.[24] The arrangement involved the prepayment principle for families only as a matter of individual agreement; whereas the insurance of entire groups, basic to current prepayment plans, was not introduced into American insurance until after 1910.[25] Just how extensive contract practice was in the United States before the Civil War is difficult to say, for one learns of it chiefly through objections voiced by professional leaders, who viewed the arrangement as demoralizing. It is doubtful if it was ever widely followed.

While on the subject of finances, one may well raise the old question: Why did the doctor's bill come last? The butcher, the baker, the candlestick-maker—all these must be paid promptly, but not the physician. Doctors themselves were none too meticulous about these matters, and many of them asked for settlement only at year's end. If not met then, the account was likely to go over another year, and so on. One of Dr. Fort's bills, noted previously, totaled $73.75. Under this was added the laconic statement: "8 yrs. int., 8 %, $47.20."

The answer to the question just posed probably lies in the conflicting traditions of the profession. From the Middle Ages was inherited a semiclerical status—physicians should be more concerned with service than with profits. Hence arose the obligation to give freely to the poor, a generosity which was rarely expected of merchants. But modern capitalism had involved physicians in a competition for income which could not be ignored and which they tried to reconcile with the service ideal in ethical codes already mentioned.[26] The public, although it recognized medical charges as an obligation, tacitly assumed that these would not be pressed. Was not the physician the friend of the family, and was not this relationship above mercenary considerations? Professionally speaking, the practitioner thus lived a double life—half benefactor, half business man—with all the difficulties this entailed. The basic problem remains today, though it seems less acute under prevailing urban forms of practice.

Close personal relations between physicians and patients reflected both scientific and social conditions. Medical science, as will be noted later, then had small bearing on therapeutics. Hence few physicians felt it necessary to give much time to current scientific developments. They were, on the other hand, under constant pressure to meet the immediate needs of their neighbors. The established practitioner in a small urban or rural environment knew the lives of his patients and the affairs of the community. There was continuity in practice, for he had brought his patients into the world and seen their fathers out of it. Other things being equal, these circumstances were of real advantage in his work. The good family physician sensed environmental and personal factors in illness: he was his own medical social worker and authority on psychosomatic relationships. Mr. X would not be sick, his doctor would remark, if he could lift the mortgage on his farm. Mrs. Y's case needed what no one could give her—the return of her lost child. The physician would use either bread pills or bitter doses if these were indicated by personal factors. Most important on the psychic side was the influence of his own authority and personality. Here he was aided by the almost

22. Shryock, R. H.: Medical Practice in the Old South, South Atlantic Quart. **29**: 173 (April) 1930.
23. Martin, R. F.: National Income in the United States, 1799-1938; cited by Stern.[9]
24. M. & S. Reporter **1**: 142, 1858. Letourneur, L. B. A.: Rapports du médicin avec la société en général, thesis, Paris, no. 155, 1821, p. 26.
25. Sickness benefits paid to members by lodges or mutual benefit societies, however, may be viewed as anticipating present group health insurance.
26. Sigerist, H. E.: Medicine and Human Welfare, New Haven, Conn., Yale University Press, 1941, p. 116.

instinctive desire of patients to have faith in the man to whom they entrusted their lives.[27]

Although there was a growing tendency after 1830 to distrust medical science in general, even the person who shared this feeling was apt to believe that his particular physician could always be of help.[28] Such faith, when misplaced, might have tragic consequences. Amid all the praise of the old family doctor it must be remembered that when incompetent he was of little use and may at times have done considerable harm. But faith in a competent man had its merits. The nice question, then as now, was how to distinguish between the able and the mediocre practitioner. Only in or near towns could there have been much range of choice, and in such locations the personal factor and sheer accident must have played major roles in the selection of family physicians. Hospital or medical school connections, often urged today as indications of professional merit, were then less significant or simply nonexistent. Graduates of certain schools seem to have had a superior reputation in special sections, as those of Harvard in New England, of Columbia and of Bellevue in New York and adjacent states, and of Pennsylvania and Jefferson in the Middle Atlantic area and the South. One authority at Yale suggested that patients, in considering a man as a prospective family physician, should engage him in conversation. If he spoke well on general matters the chances were that he was also intelligent in his practice. Beyond this there seems to have been little consideration of a problem which was so vital to the individual layman.[29]

Once having selected his physician, the American of the early nineteenth century was likely to place the greatest trust in him. Contemporary physicians might well envy the Dr. Watkins mentioned in the following note, in view of the naive confidence displayed by his patient. The latter was Miss Virginia Randolph, a granddaughter of Thomas Jefferson and an intelligent and cultivated young woman. In a letter to her fiancé in 1822 she confided that:[30]

> We have had a rather sick family lately . . . and I am on Dr. Watkins' list. He has bled me twice, and prescribed a course of calomel which I am getting safely through . . . and I expect soon to be perfectly restored. You must not expect . . . that my health is worse than it frequently is, in summer, but Dr. Watkins wishes to make a permanent cure at once of all my complaints. . . . I have no doubt that his advice is most judicious.

The cynical reader will observe that it was the "course of calomel" rather than the illness which the patient was "getting safely through," and will no doubt marvel at Dr. Watkins' determination to cure *all* her difficulties *permanently* and *at once*. Yet Miss Randolph was probably none the worse for her faith in this case, and perhaps it sustained her in later and more serious "complaints."

The successful physician was then, as now, an overworked man. In the case of the city practitioner this was a matter of many patients—particularly if he were associated with a hospital. "The average of my private patients in the late summer and fall months," wrote a physician in 1849, "is generally between 30 and 40, and to get through with these . . . I am going the round from sunrise to 9 or 10 o'clock at night." In addition, he saw daily from 60 to 70 hospital patients. "The profession," he added on another occasion, "is one which certainly requires that a man should give up almost every social enjoyment and is therefore one which a person like myself who has so keen an enjoyment of such pleasures ought never to have chosen."[31]

This does not mean that all physicians were denied social diversions, though clearly their evenings were not as free as those of other men, and they had little time for political activities or public affairs. Lawyers could run for legislature or Congress; physicians seldom had the time. In the large cities, however, prosperous physicians enjoyed the social life of medical clubs. One of the two original secretaries of the American Medical Association, en route to the organizing meeting in 1846, wrote as follows of professional hospitality in Philadelphia:

> This is a delightful House. . . . The assortment of wines, sherry, sauterne, champagne, and madeira was delicious. . . . After dinner we went to the Opera. . . . We were obliged to lose the third act, to go to Dr. Bell's Medical Club. The physicians here are very sociable amongst each other and meet three times a week at each other's houses. . . . We returned to the Opera and heard the third act and after that Dr. Clymer took me to the Club . . . I had an idea that they were all as stiff as if done up in buckram, but at this club, which is held at a large house in Walnut Street occupied solely by it, [there are] reading rooms below, card rooms and supper room on the second floor and billiard rooms on the third. I met Drs. Wm. Rush, Randolph, Biddle, Gerhard and Fox. . . . Without knowing it I played a game of five-dollar whist, but as soon as I was square I stopped playing.[32]

The country practitioner had no such opportunity for professional sociability, and such diversions as he had were those of the community at large. However, his lack of contact with colleagues was nothing like the deprivation that this seems today in isolated areas. The nature of practice was so simple that there seemed little technical need for a hospital or for professional discussions. Deliveries were made at home, and the kitchen table still seemed adequate for emergency surgical procedures. Incidentally, the introduction of anesthesia in the late forties aided surgical practice in country homes as well as in city hospitals.

The rural physician handled all phases of practice, including pharmacy and dentistry. While he saw fewer patients than did his urban colleague, he was kept just as busy by long hours spent on the road. It is not easy to picture this old routine, though much has been written about "the horse and buggy doctor." As a matter of fact, the buggy was not the only means of transportation. The physician often rode an old mare equipped with saddlebags which contained drugs and a surgical kit. He was on the road so much of the time that he could have no regular office hours: patients must take their chances on finding him at home. When a railroad reached a community, the physician would use that if possible, sometimes being extended the doubtful privilege of riding the freight cars. Occasionally he even sallied forth on a "railroad tricycle,"

27. Pusey, W. A.: A Doctor of the 1870's and '80's, Springfield, Ill., Charles C Thomas, Publisher, 1932, p. 87.
28. See North American Rev. **33**: 368, 1831.
29. Cf. Swackhamer, G.: Choice and Change of Doctors, New York Committee on Research in Medical Economics, 1939.
30. Randolph, V., to Nicholas Trist, Monticello, Va., July 26, 1822, in Trist Manuscripts, Library of Congress.
31. Shryock,[19] Arnold to his daughter Ellen, Nov. 3, 1849, p. 170.
32. Shryock,[19] Arnold to his wife, April 13, 1846, p. 166.

similar to the later handcar.[33] These devices, like runaway horses or modern automobiles, had their own peculiar risks.

One form of practice was peculiar to the days before the Civil War. This was the care of slaves. On southern farms of moderate size practice was usually in the hands of the mistress or of overseers, who were guided by tradition and by family medical manuals. Dr. Samuel Cartwright referred in 1850 to "the practice among 3 millions of people [Negroes] that the overseers have mostly got."[34] Even slaves themselves occasionally acquired a reputation as folk practitioners.

It was argued in defense of slavery that it gave the master a financial interest in preserving the health of his "people," and this was doubtless true in the case of the more enlightened and prosperous planters. Such men would hire Irish laborers rather than put their slaves into dangerous work. If a slave died, there was a loss of several hundred dollars; if an Irishman perished, there was just one less Irishman in the world![35]

Wealthy planters would regularly employ a prominent local physician to look after their plantations. This gave slaves as good care as was then available and provided them with a rough equivalent of modern health insurance. There were certain legal aspects here which were peculiar to slavery. In cases which required surgical intervention, for example, the master's permission rather than that of the patient was required. (In one curious instance the great gynecologist Marion Sims could not secure such permission and actually purchased a patient in order to operate on her.[36]) After emancipation most of this service was lost, and the rural southern Negro now receives less medical care than do members of any other large element in the population.

Implicit in all that has been said is the fact that nearly all physicians in 1846 were general practitioners. Certain persons became especially interested in surgery, but they seldom abandoned general practice entirely. This combination of the physician and surgeon in one practitioner came down from a time of primitive colonial conditions, when differentiation would have been impractical. As a result surgery always maintained equal prestige with internal medicine, in contrast to the professional and social distinctions which had been traditional in Britain.

It is usually assumed that specialization was lacking at this time simply because there was not enough knowledge to justify it. This was indeed one factor in the situation. But there was another basic explanation. Prior to about 1830, American physicians did not usually think in terms of a localized pathologic process or of specific diseases. Speculative pathologies had dominated medical thought up to that time. All forms of illness were ascribed to one or two generalized conditions, such as impurities of the blood or the existence of excessive tension or laxity in the nervous and vascular systems. These were appealing theories, since they solved all pathologic problems at once and also opened up a royal road in therapeutics. If all illness was due, as Benjamin Rush claimed, to excessive tension in the blood vessels, then all patients could be cured by bleeding to relieve that tension. Any one could see that if the patient was bled long enough he would relax—sooner or later! Hence the physician should put no faith in the healing powers of nature but rather should interfere with nature as promptly as possible.

As long as all forms of illness were viewed as expressing a single, underlying condition, there was no occasion for specialization in practice. Every physician considered that he had the remedy that was "good for what ailed you," since by definition the same thing ailed every one. Incidentally, there was also no use for major surgical operations, for one could hardly operate on the blood or on a vague condition common to the whole vascular system.[37] Hence surgical practice remained largely a matter of fractures, amputations and other superficial emergencies, and here again there was little need for specialization.

Only when a localized, structural basis for pathology replaced the speculative systems was specialization made possible. When specific diseases were identified by correlating lesions with symptoms, the possibility of treatment by removing these lesions became obvious. Surgery was therefore beginning, by 1840, to move from the periphery toward the center of medical practice. And there was now some reason for specializing, in other fields than surgical practice, in work on a particular part or system.[38] But these new points of view, imported from Paris by returning American students after 1820, percolated slowly into average practice. Not until after the Civil War did specialization really get under way, even in the cities.[39] There was strong opposition at first, partly because of the fear that it would injure the interests of both patient and practitioner;[40] partly because in the earlier day only quacks had claimed to specialize. Dr. S. Weir Mitchell could recall the time in Philadelphia when to specialize meant to be professionally ostracized.[41]

The French school of 1800-1850 was as critical of the traditional therapeutics as it was of the older pathologies. Since it identified diseases by symptoms as well as by lesions found at autopsy, it developed methods for improving clinical observation. The stethoscope and clinical statistics are cases in point. But the use of the latter showed the ineffectiveness of bleeding, purging and the old drugs. Hence French clinicians gradually lost interest in therapeutics. How could there be an intelligent search for the causes and cures of diseases until the diseases themselves had first been found?

Medical research thereupon became nihilistic; it was focused on diagnosis rather than on treatments. The latter were viewed as useless in most cases. Oliver Wendell Holmes, on his return from Paris, informed

33. Pusey,[27] p. 39. Hertzler, A. E.: The Horse and Buggy Doctor, New York, Harper & Brothers, 1938, p. 61.
34. Cited by Fenner, E. D.: Southern Medical Reports, New Orleans, B. M. Norman, D. Davies & Son, 1850-1851, vol. 2, p. 423.
35. Phillips, U. B.: Life and Labor in the Old South, Boston, Little, Brown & Co., 1929, p. 186.
36. Shryock,[29] p. 172.
37. Sigerist, H. E.: Surgery at the Time of the Introduction of Antisepsis, J. Missouri M. A. **32**: 169, 1935.
38. Rosen, G.: The Specialization of Medicine, New York, Froben Press, 1944, p. 15.
39. The gradual transition from general practice to specialization in surgery in a small city about 1880 is described in Wheeler, J. B.: Memoirs of a Small-Town Surgeon, New York, Garden City Publishing Company, 1935, p. 176.
40. Ware, J.: Success in the Medical Profession, Introductory Lecture, Massachusetts Medical College, Boston, D. Clapp, 1851, p. 9.
41. Mitchell, S. R.: The History of Instrumental Precision in Medicine, Univ. M. Mag. **4**: 1, 1891.

the American people that if most of the medicines used here were thrown into the sea it would be all the better for mankind and all the worse for the fishes![42] What was needed, he felt, was less medical interference and a revived confidence in the healing powers of nature. This critical view affected average practice only after a long interval; indeed, Osler was still preaching it in the first edition of his textbook near the close of the century.[43]

It is true that by the fifties some younger physicians indoctrinated by French-trained professors were becoming less inclined to bleed and purge, less given to excessive medication. But most practitioners clung to traditional forms of treatment. Some of the older men, even those who taught in the better schools, were still defending venesection in the seventies.[44] This half century lag in bringing practice into harmony with research is an interesting phenomenon which has never been adequately studied.

In reaction to such professional conservatism, popular opposition developed against heroic treatments. Such was the protest against calomel (mild mercurous chloride), for example, that a large and thriving medical sect—the Thomsonian or "Botanic System"—based its appeal on the condemnation of all mineral or metallic remedies that had come into use since the days of Paracelsus. Echoes of its propaganda survive today in the advertisements of secret remedies that "contain only vegetable compounds."

Popular distrust of medical treatments was based, by 1840, on more than a dislike of unpleasant or dangerous remedies. By this time physical science was making such headway that all could appreciate it. But where were the *medical* equivalents of the steam engine or the telegraph? Medicine seemed to have fallen behind the other sciences: indeed, the leading professors of medicine now admitted that their science was of little utility. Perhaps the old medical "systems," with their speculative pathologies, were really more valuable. They had at least promised cures. So encouraged, various theorists revived the doctrine that there was one cause and therefore one cure of disease. Thus Hahnemann reduced most pathologic conditions to the itch and most treatments to the "like cures like" theory and the high dilution formulas, somewhat as Dr. Still would later reduce all pathologic processes to spinal conditions and all therapeutic practice to spinal manipulations.

Regular medicine, which had accepted such oversimplifications in Rush's day, was now more critical and would have none of them. Homeopaths and Thomsonians thereupon became the modern medical sects. They founded their own schools and journals and so contributed to the confusion in education and licensing which has previously been mentioned.[45] On the other hand, the sects probably did more than did French nihilism to create a salutary demand for milder treatments.

The more credulous or nervous folk also flocked to a whole series of minor cults which promised sure and pleasant cures. Nearly every conceivable type of humbug preyed on the people—hydropathy, chronothermalism, mesmerism, herb doctors, faith doctors, indianopathists and what you will! Meanwhile national advertising in newspapers was originated by the manufacturers of secret remedies, and more money was spent for nostrums than for prescriptions. When regular physicians protested, they were viewed merely as opposing competitors. Was not this a free country, and did not every American have an inalienable right to life, liberty and quackery? A writer in one of the journals of opinion suggested that "the public will act as an umpire" between the regulars and the sectarians, and reach its decision "after a careful perusal of the undertaker's bills."[46]

It is to be recalled that this was an age of social ferment and reform, the day of the great temperance, antislavery and women's rights movements as well as of minor utopian aspirations. "We learn from the papers," observed Dr. William Alcott in 1835, "that the American Seventh Commandment Society, the New York Female Benevolent Society, the New York Ladies' Moral Reform Society, and the Anti-Tobacco Society held their annual meetings at New York about the middle of May, and that some of these meetings were interesting."[47] In such an atmosphere it was natural that a utopian health cult should arise, advising all to maintain health by staying away from the physicians and by cultivating personal hygiene. Their views represented the logical extreme of the "trust nature" philosophy, which was preached in more moderate form by physicians who had been influenced by French nihilism. Sylvester Graham, the leader of the cult, preached during the forties the virtues of bathing, of exercise and of a whole-wheat diet. In his spare time he originated an interesting literary tradition later known by the phrase "What a Young Man Should Know." His antimedical doctrine was spread by "Ladies Physiological Reform Societies," and he is unconsciously recalled to this day in the terms "Graham bread" and "Graham crackers."[48]

The appeal for personal hygiene had some merit at a time when the Saturday night bath was a real victory for sweetness and light. But more significant was the public hygiene of the period. About the end of the eighteenth century, the medieval doctrine of contagion in epidemics was partially replaced by a revival of the classic "airs and waters" hypothesis. If epidemics originated in the local contamination of air and water, then quarantines were of little use. "Noxious Miasms" could be eliminated only by cleanups, by sanitary reform. Philadelphia led the way, after its bitter experiences with yellow fever, in constructing one of the first steam-powered water works in the country (1802) and by other municipal improvements. Bitter controversy continued, however, over whether certain diseases were spread by miasms or by contagion.

42. Holmes, O. W.: Currents and Counter-Currents in Medical Science, Boston, Ticknor & Fields, 1861, p. 39.

43. By that time such nihilism was almost out of date, for pharmacology and bacteriology already held real promise for both prevention and cure.

44. I. e., as a general procedure in many forms of illness. See, e. g., Littell, S.: Memoir of George B. Wood, Trans. Coll. Physicians, Philadelphia **5**: XXV, 1881. Shryock,[1] p. 135.

45. Some forty Thomsonian journals were published in the United States between 1825 and 1850. For bibliography of the sects, Shryock, R. H.: Public Relations of the Medical Profession in Great Britain and United States, Ann. M. Hist. **2**: 308, 1930 [notes 41-44].

46. The Medical Controversy, United States Democratic Rev. **35**: 263, 1855.

47. Boston Moral Reformer **1**: 225, 1835.

48. Shryock, R. H.: Sylvester Graham and the Popular Health Movement, Mississippi Valley Hist. Rev. **18**: 172, 1931.

Quite naturally, as epidemic diseases were more clearly identified, distinct causal factors were sought in terms of one of these theories or the other. Specific pathology led in turn to a search for specific etiology. Those who held to miasms tried to find poisons in the air peculiar to given diseases, while those who accepted contagion turned to the old theory that animalcules or micro-organisms might convey specific infections. Such American physicians as Daniel Drake of Cincinnati, J. C. Nott of Mobile, Ala., and J. K. Mitchell of Philadelphia wrote during the 1840s in support of the animalcular hypothesis.[49] Their views, based on an intelligent interpretation of epidemiologic data, represented more than mere speculation. But they failed to verify their theories by laboratory studies. Hence the pioneer work in parasitology and medical bacteriology between 1840 and 1870 was left largely to Europeans.

The failure of Americans to contribute much to medical science prior to the present century may be explained by a number of factors.[50] Notable was the indifference of a commercially-minded people to research that promised no immediate utility. Why should any one encourage studies in pathology or in physiology, if these were of no help to patients? Professional prestige here was acquired rather through a fashionable practice, which presumably aided the sick and at the same time bore the hallmark of financial success. Such a practice left small time for original investigations.[51]

It is true that a few physicians also acquired reputations through professorships and consulting practices, but this was by no means the same thing as research. These men edited European works, "putting British portraits of disease in American frames," and occasionally compiled textbooks of their own; but it is remarkable how infrequently even the famous professors made any systematic investigations. A committee of the American Medical Association reported in 1850 that only two "experimental memoirs" of any importance had appeared during the preceding year—one on poisons and one on the nervous system of the alligator![52]

While the American public was relatively indifferent to pure science, it was much concerned with applied science. Hence arose all the debate about medical practice and the search for immediate cures. Most pressing in the public mind was the fear of epidemics. Outbreaks of yellow fever had inspired the beginnings of sanitary reform by 1800, but it was the terrifying specter of cholera which during the thirties and again in the fifties aroused a real demand for protection. Meanwhile, municipal authorities were disturbed by the rising death rates of various communicable diseases and learned with interest of similar concern abroad. Since sanitation seemed as much a matter for engineers and statisticians as for physicians, laymen at first assumed leadership in the public health movement. Thus Lemuel Shattuck, onetime teacher, submitted an excellent plan for a state department of health in Massachusetts (1850), which was in part the basis for the state board eventually set up there in 1869.

A few socially-minded physicians realized, however, that public health programs needed medical as well as mathematical guidance. At the start of its career the American Medical Association appointed a committee on public hygiene, which submitted valuable reports on the sanitary—or rather, unsanitary—condition of American cities and called for remedial legislation.

Soon after these reports had been made, one of the leaders of the Northern Medical Association took the center of the national stage. It happened that an international sanitary conference was called in Paris in 1855 in order to discuss cooperation in quarantine legislation. This suggested to Dr. Wilson Jewell, of the Philadelphia Board of Health, that state quarantine laws were just as diverse in the United States as were national statutes in Europe and that Americans faced the same need for coordination. He therefore persuaded his board to call a national sanitary convention, which met in Philadelphia in 1857. Subsequent meetings were held, during the ensuing three years, in Baltimore, New York and Boston. Municipal officials and socially-minded physicians joined in discussions of quarantines, sanitary regulations and the need for pure food and drug laws. This principle of bringing laymen and physicians together for the promotion of health programs was later to be employed effectively by such voluntary organizations as the National Tuberculosis Association.

Jewell was the leading spirit in these first national sanitary conventions. That of 1860 planned to transform the meetings into a permanent national health association, but the session of 1861 was unfortunately prevented by the Civil War.[53] A national body was not actually formed until the American Public Health Association came into being in 1872. It would be interesting if more could be learned about Dr. Jewell, both in relation to his leadership in health reform and to his professional career in Philadelphia. It is ironic that a leader who felt he had inaugurated "a new era in the domain of American Science"[54] has been so largely forgotten in the annals of public health.

This brings me back to my starting point, the advent of this Association, for Jewell was one of its founders.[55] Evidently he was concerned with the problems of private practice as well as with those of public hygiene. Not all aspects of such practice have been considered here, but enough has been said to suggest that the physician of a century ago faced some problems that were peculiar to his time and others which persist today. The better physicians of 1846 would be proud of the present status of medical science and of the medical guild. Indeed they would be amazed by it all. The physician of today also should take a little pride in them, for they labored long under difficult circumstances to advance the interests of their patients and of their profession.

49. Allen, P.: Early American Interest in Etiology, Hist. J. Med. & Allied Sc., to be published.

50. Shryock, R. H.: American Medical Research: Past and Present, New York, Commonwealth Fund, 1947, to be published.

51. See the contemporary protest against this situation in Jackson, S.: Address to the Medical Graduates of the University of Pennsylvania, Philadelphia, T. K. & P. G. Collins, 1840, p. 15.

52. Fishbein, M.: History of American Medical Association, J. A. M. A. 132: 920 (Dec. 14) 1946.

53. On Jewell's leadership, see his letter to Dr. J. H. Griscom, in the Proceeds. of the Third National Quarantine and Sanitary Convention, New York, 1859, p. 3 ff. Plans for a permanent national association are noted in Proceedings and Debates, Fourth National Quarantine and Sanitary Convention, Boston, 1860, p. 8.

54. Jewell's words at the first meeting in Philadelphia.

55. A centennial history of the Association has been prepared by its president, Dr. Maurice Jacobs.

RENAL DEFICIENCY ASSOCIATED WITH SHOCK

Chairman's Address

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Many writers [1] have noted that the development of shock is accompanied by oliguria or anuria and by the retention of nitrogenous wastes, often leading to uremia if the subject survives a few days. This feature is not one of the debatable aspects of shock, yet its mechanism and significance are not adequately discussed in medical literature. It has been shown [2] that shock is accompanied regularly by severe parenchymatous degeneration, especially pronounced in the kidneys; often this produces necrosis of the tubular epithelium. The possibility that these changes are related to the functional deficiency deserves thoughtful consideration.

In some instances a state of sublethal shock continues for several days; then renal deficiency often dominates the clinical picture. The urine is dark, smoky in color and has a high specific gravity. It contains albumin, hemoglobin, débris, epithelial cells, erythrocytes and casts, both granular and hyaline; frequently the casts are deeply pigmented with derivatives of hemoglobin. These features are accompanied by progressive retention of nitrogenous wastes leading to the clinical syndrome of uremia.

The occurrence of shock is not limited to trauma, burns and the aftermath of extensive surgical intervention. Harkins [3] listed more than thirty conditions in which shock may develop. These included burns, freezing, vascular occlusions, intestinal strangulation, heat stroke, radiation burns, sunburn, bile peritonitis, visceral perforations, the effects of various drugs and poisons, venoms, peptone, anaphylaxis, transfusion reactions, metabolic intoxications and infections of fulminating severity. I have reported the occurrence of shock under these and other conditions and have collected evidence on renal deficiency associated with many of them. That evidence is too extensive for detailed review here. It included, among others, Bell's [4] report on clinical acute nephritis as distinct from glomerulonephritis, Bordley's [5] and Daniels' [6] contributions on renal effects of transfusions, Lambret and Driessens' [7] studies on surgical shock, the reports of Helwig [8] and Boyce [9] on uremia associated with so-called "liver death," Christophe's [10] observations on acute nephritis as a regular feature of burns, both clinical and experimental, Bywaters' [11] observation on uremia from compression of limbs and the review of Jeghers and Bakst [12] on extrarenal uremia.

The reported conditions under which acute uremia developed independent of glomerulonephritis included intestinal obstruction, toxic jaundice, hepatorenal syndrome, extensive surgical intervention, intestinal perforation, poisoning with phenobarbital, cinchophen and other drugs, streptococcic cellulitis and other severe infections, diabetic coma, Addison's disease, anaphylactic reactions, transfusion with incompatible blood, acute pancreatitis, cerebral lesions such as abscess or hemorrhage, sublethal shock from trauma as in the crush syndrome and burns.

I have confirmed the pathologic features described by others under the conditions mentioned. Grossly the kidneys may be of normal size or moderately enlarged; usually they are soft, indicating edema. The capsule strips with unusual ease; the stellate veins in the cortical surfaces are engorged. The color varies, depending on relative degrees of congestion and of parenchymatous changes. Cloudy swelling causes varying degrees of pallor; but when congestion predominates the color is deep red. Hemorrhagic streaks are seen in the medulla, and the cortical markings frequently are obscured. Petechiae may be seen in the parenchyma and in the pelvic lining.

The microscopic picture presents varying degrees of changes within a regular pattern. Usually hyperemia of the glomerular tufts and of the intertubular vessels is conspicuous. Sometimes capillary hemorrhages are present. Frequently amorphous material is seen in the capsular spaces. The epithelium of the convoluted tubules shows acute degeneration ranging to necrosis. When death occurs after several days, the cells are low or cuboidal in shape and the lumens are wide. The cytoplasm is granular; more advanced degeneration is indicated by vacuolization of the cytoplasm or by disintegration of the cells. Nuclear changes, especially karyolysis and hyperchromatism, are conspicuous.

Usually these changes are more pronounced in the upper segment, sometimes in the lower, but in general all portions of the convoluted tubules are affected. Plugs of débris often form in the lumens of the lower segments because such débris, originating in the upper portion of the nephron, collects in the narrow lumen of the lower portion. Hyaline droplets may form in the cytoplasm when degeneration is advanced; the lumens may contain masses of this deep red material when the cells containing it have disintegrated. Hyaline, granular and, sometimes, pigmented casts, débris, erythrocytes, nuclei and masses of desquamated epithelial cells occur in the collecting tubules. Edema is a varying feature, more pronounced in the medullary than in the cortical zones.

A widely known instance of this condition is the crush syndrome reported by Bywaters. Persons in buildings destroyed by explosions often were pinioned for many hours beneath masonry and débris. Some had no external wounds or broken bones, but extensive areas of flesh had been affected by continued heavy pressure. When rescued, these victims showed no serious effects except numbness and paralysis of the

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Permission for the publication of data from the Army Institute of Pathology was granted, but sanction for the author's interpretations was disclaimed by the Medical Department of the United States Army.

Read in a symposium on "Renal Insufficiency in the Shock Syndrome" before the Section on Pathology and Physiology at the Ninety-Fifth Annual Session of the American Medical Association, San Francisco, July 3, 1946.

1. Whipple, G. H.; Smith, H. P., and Belt, A. E.: Relationship of Blood Proteins to Shock, Am. J. Physiol. **52**: 72, 1920. Aub, J. C., and Wu, H.: Chemical Changes in the Blood in Traumatic Shock, ibid. **54**: 416, 1920-1921. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1931, vol. 1, p. 292; vol. 2, p. 590. Blalock, A.: Principles of Surgical Care, Shock and Other Problems, St. Louis, C. V. Mosby Company, 1940, p. 94.

2. Moon, V. H., and Kennedy, P. J.: The Pathology of Shock, Arch. Path. **14**: 360 (Sept.) 1932. Moon, V. H.: Shock: Dynamics, Occurrence and Management, Philadelphia, Lea & Febiger, 1942, chap. 9 and 15.

3. Harkins, H. N.: Study and Management of Traumatic Shock, Surgery **9**: 231, 1941.

4. Bell, E. T.: Pathology and Pathogenesis of Clinical Acute Nephritis, Am. J. Path. **13**: 497, 1937. Renal Diseases, Philadelphia, Lea & Febiger, 1946, pp. 254-264.

5. Bordley, J., III: Reactions Following Transfusions of Blood with Urinary Suppression and Uremia, Arch. Int. Med. **47**: 288 (Feb.) 1931.

6. Daniels, W. B.; Leonard, B. W., and Holtzman, S.: Renal Insufficiency Following Transfusion, J. A. M. A. **116**: 1208 (March 22) 1941.

7. Lambret, O., and Driessens, J.: Les modifications humorales post-opératoires, J. internat. de chir. **2**: 223, 1937.

8. Helwig, F. C., and Schutz, C. B.: A Liver-Kidney Syndrome, Surg., Gynec. & Obst. **55**: 570, 1932.

9. Boyce, F. F., and McFetridge, E. M.: So-Called "Liver Death," Arch. Surg. **31**: 105 (July) 1935.

10. Christophe, L.: La mort des brûlés, Paris, Masson & Cie, 1939.

11. Bywaters, E. G. L.: Effects on Kidney of Limb Compression, Brit. M. J. **2**: 884, 1941.

12. Jeghers, H., and Bakst, H. J.: The Syndrome of Extrarenal Azotemia, Ann. Int. Med. **11**: 1861, 1938.

compressed limbs. Presently signs of shock developed, but these were counteracted by transfusions of blood and of plasma. Even though satisfactory circulation and blood pressure were maintained, renal function did not return and the victims died of uremia.

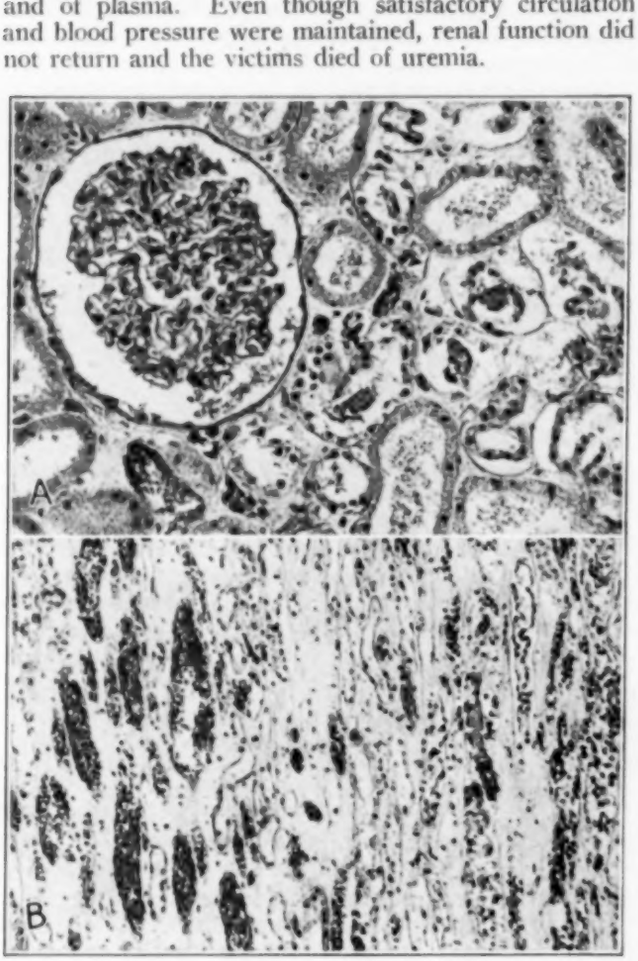

Fig. 1 (case 1).—Photomicrograph (*A*) of renal cortex after traumatic shock. Note the granular material in the capsular spaces and in the lumens. The epithelium is cuboidal and the lumens wide; the cells show parenchymatous degeneration, and some are necrotic. The nuclei are hyperchromatic; some have disappeared. There are casts and desquamated cells in the lower segments of the tubules (Army Medical Museum, negative 86832). *B*, medullary portion of the same kidney as *A*. Desquamation of lining, blood casts, pigmented casts and edema are shown (Army Medical Museum, negative 86839).

The kidneys in such cases presented the picture just described. It was considered that the renal deficiency was due to mechanical blockage of the collecting tubules by plugs of pigmented débris. Were this the case, the majority of the tubules should show stoppage, and the gross size of the kidneys should be greatly increased. In sections from a patient with a typical case of shock syndrome furnished by Dr. Bywaters, only a minority of the collecting tubules were obstructed. The histologic features were identical with those resulting from surgical shock or from low atmospheric pressure (figs. 1 and 2).

Numerous instances of renal effects were found during a study of shock in the records and specimens collected by the Army Institute of Pathology. A few typical cases will be cited.

REPORT OF CASES

Case 1.—A man aged 39 suffered lacerations, contusions and multiple fractures of the femur, ankle and ribs. He was in profound shock on admission to the Station Hospital; the pulse and blood pressure could not be determined. Transfusions of blood and fluids given intravenously were ineffective; he died of progressive circulatory failure thirty hours after the accident. The kidneys (fig. 1) show all the characteristic changes previously described, although the subject died before renal deficiency became advanced.

Case 2.—Resection of rectal carcinoma, performed under spinal anesthesia on a man aged 59, "was followed immediately by marked shock from which he did not entirely recover." Repeated transfusions of blood and of plasma did not restore circulatory efficiency. The blood pressure fluctuated between 60 and 100 diastolic. Urination was almost completely suppressed. The nonprotein nitrogen and creatinine rose progressively, and death occurred on the fourth day. The clinical diagnosis was uremia secondary to severe postoperative shock.

The kidneys weighed 210 and 190 Gm.; the subcapsular vessels were engorged; the cortex was thickened, opaque and pink. The tubular epithelium showed granular degeneration, decided vacuolization and beginning necrosis, which involved chiefly the upper segment. The tubules contained débris and desquamated epithelium, and there was moderate edema of the stroma.

The literature contains many observations on the renal effects of burns, so I shall give only 1 additonal example:

Case 3.—This young man's clothing was saturated with gasoline and ignited. He sustained first, second and third degree burns of the entire body. Treatment for shock with morphine, plasma and dextrose-saline injections was begun at once; the burned areas were debrided, and pressure dressings were applied. The urine was scanty and dark; it contained albumin, red blood cells and blood casts. On the fourth day the total output was only 200 cc. of coffee-colored urine. No chemical determinations were made. Death occurred at the end of the fourth day.

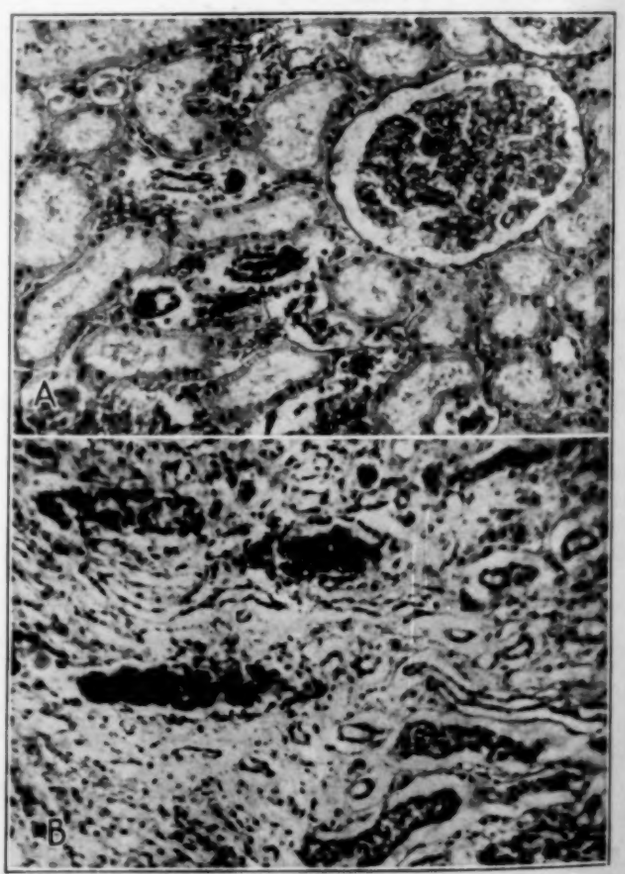

Fig. 2 (case 7).—Photomicrograph (*A*) of renal cortex after shock from low atmospheric pressure. Pronounced hyperemia, granular material in capsular spaces and in lumens, hyperchromic nuclei, degeneration and necrosis of epithelial cells, blood and pigmented plugs within the tubules are seen (Army Medical Museum, negative 86841). *B*, renal medulla from the same case. Desquamation of the lining and cellular, hyaline and pigmented casts are shown.

The visceral findings at necropsy were those usually seen after death from burns. The kidneys weighed 200 and 240 Gm.,

respectively; they were soft, swollen and grayish pink; the cortical markings were obscure, and the superficial vessels were engorged. Amorphous material was present in the capsular spaces; the tubular epithelium showed advanced degenerative changes; it was low cuboidal in form, and the lumens contained débris and casts consisting of red blood cells, hyaline and dark

Fig. 3.—Photomicrograph (*A*) of renal cortex after death from burns. Pronounced hyperemia, erythrocytes in capsular space, epithelial degeneration and necrosis, débris and pigmented plugs within the lumens are shown (Army Medical Museum, negative 89865). *B*, photomicrograph of renal medulla after shock from intestinal obstruction plus surgical intervention. Hyaline and pigmented casts, erythrocytes within the lumens and desquamation of the lining are shown (Army Medical Museum, negative 86830).

brown pigment. The color of the pigment is of particular interest because the patient had not received transfusions of blood, sulfonamide compounds or other drugs except penicillin. Evidently the occurrence of pigmented casts is not dependent on transfusion or on sulfonamide therapy.

It is commonly known that severe infections of various types often disturb renal function. This seems to depend on the severity rather than on the species of the infection. Numerous accounts of febrile albuminuria may be found; only occasionally did the authors of such articles recognize a relationship to shock. The following is an instance.

CASE 4.—Severe illness developed in this man, with moderate leukocytosis and a temperature of 103 to 104 F. He was treated with sulfonamide drugs and penicillin without effect. The blood pressure declined to 80 systolic and 60 diastolic. He became anuric, and uremic crystals covered the cutaneous surfaces. The nonprotein nitrogen rose to 288 mg. and the creatinine to 6.6 mg. per hundred cubic centimeters of blood, on the eleventh day of illness. He died in coma on the twelfth day. From the character of the illness and the postmortem observations a provisional diagnosis of systemic tularemia was made.

The necropsy revealed numerous scattered nodules in the lungs and spleen. These contained necrotic areas, fibrin and monocytic infiltration such as occur in tularemic inflammation. The spleen and liver were enlarged, the viscera were hyperemic. The kidneys were moderately swollen and contained capillary hemorrhages. There was extensive degeneration and necrosis, especially severe in the proximal portion of the convoluted tubules. The lumens contained débris and casts, both hyaline and granular. These features were of the same order as those seen in renal deficiency from other causes.

It is well known that either hyperthermia or heat stroke may cause circulatory collapse; in some instances the subject may survive several days in a state of sublethal shock accompanied by renal deficiency. The following case reports are instances.

CASE 5.—Circulatory collapse developed in this man after six hours at 107 F. in hyperthermic treatment for gonorrhea. On his removal from the cabinet the blood pressure fell to 50 systolic and 30 diastolic. The pulse rate was 156, and the urea nitrogen of the blood was 22 mg. per hundred cubic centimeters. There were oliguria and albuminuria, and the urine contained erythrocytes and casts. Supportive treatment was inffective, the circulatory deficiency continued and the blood urea nitrogen level rose to 77 mg. per hundred cubic centimeters. Death came ninety-eight hours after the therapeutic hyperthermia.

As is usual in such cases, the visceral findings were those characteristic of secondary shock. The kidneys, weighing 240 and 250 Gm., were swollen and reddish brown. The cortical markings were obscure, and the pelvic lining contained petechial hemorrhages. Microscopically, both cortex and medulla were hyperemic. The tubular epithelium was severely degenerated; the lumens were wide and contained débris and desquamated cells; the collecting tubules contained casts and many erythrocytes. The stroma was edematous.

CASE 6.—In this man there developed nausea, vomiting, weakness and circulatory deficiency while he was working in excessive heat. Other observations were a rapid pulse rate, low blood pressure and hemoconcentration. The following day the urine was scanty; it contained albumin, red cells and casts. The circulatory deficiency continued despite the administration of plasma and saline fluids intravenously. The blood urea level rose progressively, and death occurred six days after the collapse. Clinically this case was designated as heat exhaustion. Pronounced visceral hyperemia, serous effusions, edema, petechial hemorrhages and severe parenchymatous degeneration were revealed at necropsy. The renal observations were similar to those in the preceding case, except that tubular necrosis was more advanced and there were more casts in the lumens.

In military aviation at high altitudes circulatory collapse resembling surgical shock often developed even though oxygen was adequately supplied. "The most dangerous aspects are seen after a latent period of one to six hours, resulting in a full-blown vicious circle of peripheral circulatory insufficiency, tissue anoxia and marked hemoconcentration."[13] I had the opportunity to examine the records and material in 5 such cases. The following is a typical example.

CASE 7.—This man was undergoing a test in the low pressure chamber at a relative altitude of 38,000 feet when pains and distress developed. After gradual recompression he seemed drowsy and incoherent, and he was put to bed immediately. The pulse rate then was 80, and the blood pressure, 110 systolic and 80 diastolic. Severe circulatory deficiency developed four hours later; the pulse could not be obtained, the hematocrit reading had risen to 64.9, the patient was sweating and apparently was in profound shock. He was given plasma and isotonic solution of sodium chloride intravenously and oxygen by inhalation. Severe hyperpyrexia developed like that which accompanies heat stroke; the rectal temperature rose to 108.4 F. The urine was scanty and contained albumin 4 plus. No blood chemical tests were made. Despite the administration of intravenous fluids and other measures the circulatory deficiency

13. Physiology of Flight, Air Forces Manual 25-2, United States War Department, Army Air Forces, 1945, pp. 17 and 36.

persisted; death came forty-five hours after the patient's removal from the low pressure chamber.

The necropsy observations were the same as those seen in secondary shock from other causes. Microscopic examination of the kidneys revealed the identical pattern of changes seen in the crush syndrome or after transfusion with incompatible blood. It is highly significant that numerous pigmented casts were found, although no transfusions and no therapy with sulfonamide compounds or other drugs had been given.

Space is lacking for details of other instances. These would include intestinal obstruction, peritonitis, perforations of viscera, coronary occlusion, radiation sickness, toxic jaundice, transfusion reaction, anaphylaxis, poisoning with various chemicals, reaction to arsenical therapy, overdoses of sedatives and of sulfonamide drugs, diabetic crises and others. Each of these conditions, if its severity is maximal, will result in the syndrome of shock leading to death. Each of them, when less severe, may cause the syndrome of uremia leading to delayed death. The morphologic changes in the kidneys are of the same pattern, regardless of the causative condition.

Several factors may operate to cause degeneration and necrosis of the tubular epithelium. Poisons, as mercury, arsenic and others, are believed to damage the cells directly. Toxins and other products of bacterial metabolism may have a similar action. Many believe that toxic factors are concerned in shock from trauma or burns. In open contaminated wounds bacterial metabolic agents are an additional factor. Bile and other metabolic agents in abnormal concentration may be injurious to parenchymatous cells. Hence, in cases of poisoning, infections, contaminated wounds and metabolic intoxications, it is possible that the agents mentioned may be responsible for the cellular damage. However, exactly the same pattern of degenerative changes develop in shock from anaphylaxis, transfusion with incompatible blood, asphyxia and anoxia of various types, coronary occlusion, heat stroke and low atmospheric pressure. The effects in these conditions cannot be explained satisfactorily as due to toxic agents. One concludes that other factors, such as neural influences or anoxia play an important role. The degeneration and necrosis of tubular epithelium probably do not result from a single cause but from several causes which operate in varying combinations.

Because of the diverse conditions of its development, the frequency of this renal disorder is high. My observations confirm those of Jeghers and Bakst [12] that "azotemia is much more commonly due to the mechanisms discussed than to organic renal disease." The patient may die of circulatory or renal failure or from their combined effects. When he survives for a time, the oliguria may suddenly give place to polyuria. This is regarded as a favorable sign, indicative of the return of renal function and the beginning of convalescence. When recovery occurs the tubular epithelium regenerates and there are no residual alterations, either morphologic or functional, in the kidneys.

Several explanations have been proposed for renal deficiency associated with sublethal shock. It has been suggested that glomerular filtration, which depends partly on capillary blood pressure, is decreased because of the low arterial pressure which accompanies shock. Reduced filtration would reduce renal elimination correspondingly. However, the blood pressure is not regularly below normal; this syndrome frequently develops even though the blood pressure is maintained at adequate levels by intravenous therapy.

Again, it has been suggested that the glomeruli are unable to produce a filtrate from blood which is above normal concentration. This factor may be of importance when hemoconcentration is pronounced. However, renal insufficiency is present often in the absence of hemoconcentration; in many cases reported, uremia was severe but the blood was not concentrated.

Several have suggested that the deficient excretion of urine is due to mechanical blockage of the tubules by plugs of débris. If this were the chief factor in reducing renal elimination, the kidneys should be swollen beyond the maximum size recorded in any such cases. Also, I have never seen an instance in which as many as 20 per cent of the tubules were obstructed by casts. The remaining 80 per cent of unobstructed tubules should be adequate to maintain renal elimination within physiologic limits.

Richards' [14] investigations on renal function provide an acceptable explanation for renal deficiency associated with damage to the tubular epithelium. They showed that degeneration of the epithelium reduced its function of selective absorption, by which the glomerular filtrate is concentrated during its passage through the convoluted tubules. When these were seriously damaged they allowed the entire filtrate to be resorbed into the circulation by a process of diffusion. This produced decided oliguria or anuria by the interruption of renal secretion. Without attempting a final explanation for renal functional deficiency associated with shock, I lean toward the interpretation suggested by Richards.

This form of renal disease is not new; it was described by internists fifty years ago under such terms as acute nephritis, acute Bright's disease and acute parenchymatous nephritis. Osler [15] cited poisoning with various chemicals and drugs, diphtheria, influenza and other severe infections, surgical operations, trauma and burns among the conditions of its occurrence. The clinical signs included oliguria, albuminuria, hematuria, casts, dark or smoky urine having a high specific gravity and uremia.

Pathologists contemporary with Osler (Ziegler,[16] Kaufmann,[17] Coplin,[18] Adami,[19] Delafield and Prudden [20]) described the chief morphologic features, as given in a preceding paragraph, under such terms as acute tubular nephritis, acute parenchymatous nephritis, desquamative or degenerative nephritis and acute Bright's disease. The conditions of its occurrence were those given by the internists. Both Adami and Ziegler stated that it may be impossible to distinguish between cloudy swelling and this form of nephritis.

When the present terminology of renal disease was proposed, acute parenchymatous nephritis or tubular disease had been dropped from the list; hence both clinicians and pathologists have lost sight of its importance. This terminology is deficient in that it provides no name for this entity. Such terms as acute tubular disease, tubular nephrosis or toxic nephrosis would indicate this condition adequately.

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18. Coplin, W. M. T.: Manual of Pathology, Philadelphia, P. Blakiston's Son & Co., 1911, pp. 656-657.
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SUMMARY

Oliguria or anuria occurs regularly during the development of secondary shock. Deficiency of renal function becomes a prominent feature if a state of sublethal shock persists for a time.

This deficiency is manifested clinically by oliguria, albuminuria, hematuria, casts and the progressive retention of nitrogenous wastes often terminating in uremia.

The pathologic renal features include hyperemia and edema, parenchymatous degeneration ranging to necrosis of the tubular epithelium, desquamation of the lining, débris, and hyaline, granular and sometimes pigmented casts in the lumens.

These changes may result from toxic or from anoxic effect on the renal epithelium. This form of renal disorder is of common occurrence because of the various conditions which may cause shock. If the causative condition is abated, recovery may follow. In such cases the cells regenerate and no permanent structural changes result.

This renal disorder formerly was known to pathologists and clinicians as acute parenchymatous, desquamative or acute tubular nephritis. It is not discussed adequately in recent medical treatises.

THE RENAL LESIONS IN EXTENSIVE CUTANEOUS BURNS

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The literature dealing with the pathologic anatomy of the kidneys following severe cutaneous burns is extensive. Descriptions of the gross lesions were published by Cumin [1] in 1823 and by Long [2] in 1840, but these are chiefly of historical interest. The modern concept of the renal changes associated with burns began with Wertheim's [3] description in 1867 of his findings in dogs, supplemented in 1868 by study of a severely burned patient. Utilizing the observations of Schultze [4] (1865) on the degenerative changes in red blood cells on a heated microscopic stage, Wertheim described crystals of blood pigment in the renal tubules in both his experimental animals and patients. The parenchymatous changes he referred to as a form of "Bright's kidney."

The presence of hemoglobin in the renal tubules after severe burns was confirmed by Ponfick [5] (1876), Fraenkel [6] (1889) and others. Ponfick also described "intensive congestion of the larger renal arteries, vasa afferentia and glomerular loops, with red blood cells, as well as a moderate fatty infiltration and hyaline casts in the convoluted tubules." Fraenkel suggested that the plugging of the renal tubules with blood pigment might be responsible for the oliguria and renal changes in the presence of serious burns.

Welti [7] (1889) found scattered hyaline and granular thrombi in the arteriolae rectae and other renal vessels of rabbits burned with hot water. Similar thrombi were produced experimentally by Silbermann [8] (1890) and were recorded in human necropsies by Bardeen [9] (1897), Weiskotten [10] (1919), Vogt [11] (1929) and others.

Inflammatory changes in the tubules of the cortex, with the formation of an exudate, were demonstrated by Schjerning [12] (1884). Wilms [13] (1901) and Marchand [14] (1908), however, denied the existence of a nephritis due to burns, though they both noted the degenerative changes in the tubules reported by earlier workers; and Marchand asserted, in agreement with Wertheim, that these changes were due to the excretion of hemoglobin. Dohrn [15] (1901) was able to find pathologic lesions of the kidneys attributable to burns in only 3 of 9 necropsies which he reported.

In a study of the internal lesions of patients dying of superficial burns, McCrae [16] (1901) noted an endothelial hyperplasia in the glomerular capillaries. In 3 cases recorded by Raysky [17] (1910) the alterations in the kidneys were of varying intensity. In 1 only slight changes in the glomeruli were present, while in another there was a diffuse necrosis. In the remaining case, which was intermediate between the others in severity, there was an infiltration of round cells, "chiefly localized about the glomeruli," but without glomerular destruction. Heyde and Vogt [18] (1913) observed an accumulation of lipoids in the interstitial tissues of the kidneys, and Zinck [19] (1938) found a high grade edema in the large renal vessels and calcium deposits in the tubular epithelium.

Moon and Kennedy [20] (1932), studying the shock syndrome, demonstrated "dilatation and engorgement of the capillaries and venules," including those of the kidneys and lungs, and increased "permeability of the capillary walls, as indicated by petechial hemorrhages and effusion into serous cavities." In his subsequent extensive work on the pathologic anatomy of shock,

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Because of lack of space this article is abbreviated in THE JOURNAL by omission of part of the illustrations.

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Moon indicated the many points of similarity between "burn shock" and shock due to other causes.

In 2 patients who died five hours and five days, respectively, after extensive burns, Davis [21] (1940) found a "congestive hemorrhagic and edematous visceral appearance," which he interpreted as typical of the shock syndrome. Olson and his co-workers [22] (1944), however, were able to differentiate between burn shock and tourniquet and hemorrhagic shock in dogs on the basis of the renal output following infusions of saline solution. In the unburned animals renal output was increased by the infusion, but no such change was observed in those suffering from shock incident to burns.

The most extensive clinicopathologic correlations in cases of burns are those of Christophe [23] (1939). On the basis of detailed clinical and autopsy records of 164 burned dogs he postulated the presence of an "acute glomerulonephritis" characterized clinically by: (1) oliguria with albuminuria and casts, or anuria, for twenty-four to forty-eight hours; (2) progressive rise in the nonprotein nitrogen and blood urea nitrogen;

TABLE 1.—*Summary of Renal Changes Associated with Extensive Burns*

I. Exclusively early changes (first 72 hours): hemoglobin casts in the tubules, bilateral cortical necrosis

II. Exclusively late changes (after 72 hours): acute interstitial nephritis, metastatic abscesses

III. Changes seen both early and late:

1. Degenerative changes in the tubules
   (a) Albuminous degeneration, vacuolar degeneration, and simple necrosis, particularly of the proximal convoluted tubule, and Henle's loop, less frequently of the distal convoluted tubules
   (b) Degenerative fatty infiltration of the tubules
2. Congestion of the blood vessels with erythrocytes
3. Interstitial and submucosal hemorrhages
4. Thrombi: hyaline in the glomerular loops, granular in the larger vessels
5. Infarcts: hemorrhagic and anemic
6. Glomerular endothelial hyperplasia
7. Hyaline, granular and hemoglobin casts
8. Interstitial edema

This recapitulation is based on the observations of previous investigators.

(3) lowering of plasma osmotic pressure and reversal of the albumin-globulin ratio; (4) decrease in total blood volume; (5) decreased alkaline reserve and p_H of the blood; (6) decreased plasma chlorides, and (7) increased blood cholesterol. The renal lesions were described by Christophe as identical with those of "acute glomerulonephritis," consisting in extensive glomerular congestion and tubular degeneration. He asserted that there is a toxic principle in the blood returning from the burned area; that in the first ten hours after the burn this principle is absorbed, and that to it, and secondarily to its effect on the brain, the visceral lesions are due.

A similar attempt to correlate the clinical and pathologic data was made by Goodpastor and his associates [24] (1946) in 47 patients with severe thermal burns, some of them victims of the Cocoanut Grove disaster of 1942. In 20 patients definite morphologic changes in the kidneys were demonstrated at necropsy. In addition to necrosis and the presence of pigment and cellular casts in the tubules, these writers reported in some instances a regeneration of the tubular epithelium, which they interpreted as "indicative of repair of tubular necrosis."

Brown and Crane [25] (1943) reported a case of bilateral cortical necrosis of the kidneys in a woman who died the sixth day after receiving severe burns involving 40 per cent of the body surface. The renal vascular damage in this case—probably the most extensive to be recorded in the literature—apparently coincided with the period of deep shock, the first forty-eight hours after the burn occurred.

Bell [26] (1946) described a vacuolar degeneration of the epithelium of the proximal convoluted tubules. Other important contributors to the literature on the renal pathology in burns include Simonart [27] (1937); Wilson, MacGregor and Stewart [28] (1938); Erb and associates [29] (1943); Baker [30] (1945); Gibson [31] (1945); Pack [32] (1926), and Shen, Ham and Fleming [33] (1943).

A brief recapitulation of the essential findings of previous workers on the renal changes associated with extensive burns is presented in table 1.

OBSERVATIONS ON CURRENT STUDIES

The present report is based on a study of 84 experimentally burned dogs and 20 cases in man. The experimental observations were made at the Henry Ford Hospital on a series of animals which has served as a basis of previous papers on other aspects of thermal burns (Hartman,[34] 1941, 1943 and 1946). Data on 14 of the human cases were obtained from the autopsy files of the Department of Pathology of the University of Michigan, while data on the other 6 came from the Henry Ford Hospital.

On the trunks of dogs previously anesthetized with morphine and chloroform, second and third degree burns were inflicted by steam jet in 12 instances and by Bunsen burner in 72. The burns were of uniform depth and extent, comprising about 60 per cent of the body surface. The animals were kept under morphine sedation, and dressings of various types (tannic acid, tannic acid-silver nitrate, ferric chloride, boric acid ointment and petrolatum) were continuously applied. As these dressings appeared to have no influence on the renal changes, further reference will not be made to them. In a few instances treatment with penicillin was given; although this seemed to retard infection and thus prolonged life; it was apparently without

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effect on the pathologic process. The dogs were observed until death, and autopsies were performed immediately. A few moribund animals were killed.

The renal observations in the animals and the human beings are summarized in table 2.

Vascular Congestion; Hemorrhages; Infarcts; Pyelonephritis.—Congestion of the renal vessels was present in at least 70 per cent of the human material and 75 per cent of the experimental animals. It was found to be greatest in the outer medulla and cortex and to involve chiefly the capillaries and venules.

Hemorrhage was not frequently observed. When it occurred independent of infarcts, it was usually in the outer zone of the medulla. Here, according to von Möllendorff [35] (1930), there are groups of fifteen to thirty small blood vessels (capillaries, arterioles, venules), and it is about these congested vessels that hemorrhages were most commonly seen, though they occurred also in the loose connective tissue beneath the pelvic mucosa.

TABLE 2.—*Renal Observations in Extensive Cutaneous Burns*

	20 Cases in Human Beings			84 Cases in Dogs		
	Present	Doubtful	Absent	Present	Doubtful	Absent
Venous and capillary congestion	14	5	1	63	9	12
Albuminous degeneration and simple necrosis of the proximal convoluted tubule	18	1	1	66	5	13
Albuminous degeneration and simple necrosis of Henle's loop	14	2	4	45	2	37
Vacuolar and necrotic changes of the collecting tubules	3	0	17	25	1	58
Necrosis and débris in Bowman's capsule	7	0	13	25	3	56
Glomerular endothelial hyperplasia	3	3	14	17	1	66
Acute interstitial nephritis	1	..	19	34	..	50
Pyelonephritis	3	..	17	8	..	76
Infarcts	3	..	17	8	..	76
Hemorrhages	4	..	16	13	..	71
Casts	7	..	13	9	..	75

Glomerular congestion was of much the same degree as that in the capillaries except in the presence of small contracted glomeruli or glomerular endothelial hyperplasia.

No hyaline or granular thrombi, such as those produced experimentally by Welti (1889), Silbermann (1890) and Simonart (1937) were seen in our materials. A possible explanation for this lies in the duration of the burning. Their animals were burned for much longer periods than ours, and it may be that the prolonged heat bears a direct relation to the production of thrombi. Septic bacterial emboli were present in 8 of our dogs and 3 of the human cases, with resultant hemorrhagic infarcts and pyelonephritis.

Albuminous Degeneration.—The albuminous degeneration and simple necrosis of the proximal convoluted tubules and of the ascending thick segments of Henle's loop did not differ from these changes as described by others, from Ponfick (1876) to the present day.

Presumptive evidence of regeneration of the tubular epithelium was seen in 29 of the 79 dogs that lived more than two days after burning. Such evidence consisted in (*a*) the presence of necrosis of the tubular epithelium, usually manifested by coarse eosinophilic debris in the lumen, without the formation of casts, and (*b*) new, well-defined low tubular epithelial cells with normal staining qualities. No mitotic figures such as those described by Goodpastor and his associates [24] (1946) and shown in their photomicrographs were encountered. While we have accepted the criteria previously noted as presumptive evidence of regeneration, we realize that they constitute only a doubtful basis for such an assumption. In the proximal convoluted tubules the medial border of the epithelium is sharply defined, and neither the brush border of the normal cell nor the frayed-out border of the necrotic cell is seen. It is extremely difficult, if not impossible,

Fig. 1 (case 3).—Photomicrograph of renal cortex (medium power). The survival time was twelve hours. Note the albuminous degeneration and necrosis of the proximal convoluted tubules in contrast to the glomeruli, which appear normal.

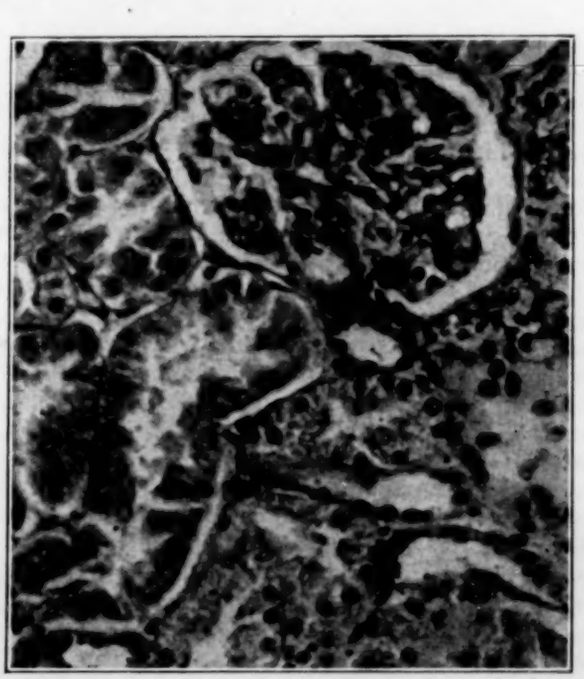

Fig. 2 (case 3).—Photomicrograph of renal cortex (medium power). Note the albuminous degeneration and necrosis of proximal convoluted tubules.

35. von Möllendorff, W.: Harn und Geschlechtsapparat, Berlin, Julius Springer, 1930, pt. 1, pp. 253-255.

to distinguish a regenerated epithelial cell from epithelial cells that have simply survived the early phases of the burn. Tilp [36] (1925), who has written extensively on "intracanalicular regeneration" of tubular epithelium, has stated that the process is characterized by the appearance of mitotic figures, the presence of

Fig. 3.—Photomicrograph of the medulla (inner zone) of the kidney of dog 3280 (medium power). The survival time was eleven days. Note the hyaline casts and the vacuolar changes in the collecting tubules.

new epithelium and the appearance of epithelial multinucleate giant cells.

Vacuolar and Necrotic Changes in the Tubules.—Vacuolar degenerative changes in the proximal convoluted tubules were seen in 3 of the dogs but were not present in any of the human cases. In no instance were they of the extreme degree shown by Bell [26] (1946) in his photomicrographs. The vacuolar and necrotic changes in the collecting tubules were principally confined to the medullary rays in the cortex. The vacuoles were of various sizes. Many filled the entire cytoplasmic portion of the cell. In such cells the nuclei were pyknotic. The cell borders were strikingly clear as prominent eosinophilic lines. The cytoplasm, however, was so modified that only a small amount of eosinophilic dust was seen in the large vacuoles. In the areas of milder change the cells had the appearance of hydropic degeneration; in other areas they appeared necrotic. These vacuolar and necrotic changes in the collecting tubules were found in 25 animals, 18 of which were in the first nine day survival group.

Similar changes were recorded in 3 of the human cases. The cause of this vacuolar change is obscure, but similar necrotic changes have been produced in the proximal convoluted tubules of rabbits by repeated injections of sucrose and dextrose (Helmholtz,[37] 1933). It is of interest that in 1 instance (case 10), that of a child aged 2½ years who showed hydropic degeneraion of the proximal convoluted tubules at necropsy, 100 cc. of a 25 per cent solution of dextrose had been administered two hours before death.

Necrosis and Débris in Bowman's Capsules Casts.—Many of the degenerating tubules and capsules of Bowman were observed to contain eosinophilic amorphous débris, but true casts were infrequent. Three patients who died within seventy-two hours after receiving their burns showed casts of erythrocytic débris. Hyaline casts were also infrequent, being observed in only 9 dogs and 7 human cases. A few fresh eosinophilic hyaline casts were noted in distended collecting tubules, and in 1 instance numerous older, partially calcified, hyaline casts were present in undistended tubules. This was the only evidence of calcification in the kidney which was encountered in our study. Extensive calcification of the renal tubules after burns was reported by Zinck [19] (1938) and attributed by him to hypochloremia.

Fatty Infiltration.—Degenerative fatty infiltration of the kidney was noted in 6 of 14 autopsies in which fat stains were used. The small round orange globules of fat, stained with sudan III, were just visible at the base of the tubular epithelial cells. In some of these cases there was an associated fatty degenerative infiltration of the heart and liver. This change was considered to be due to the same factors as those which produce albuminous degeneration and simple necrosis, i. e., anoxia and metabolic poisons. In some of the cases reported here the process was so mild as to appear reversible.

Glomerular Endothelial Hyperplasia.—Glomerular endothelial proliferation was infrequent and, except in a few cases of old infected burns, was of a minimal and even questionable nature. In no case was the endothelial proliferation great enough to occlude the capillary lumens. Necrotic changes were also infrequent and, when they did occur, were usually attributable to embolic infections and postmortem change. We agree

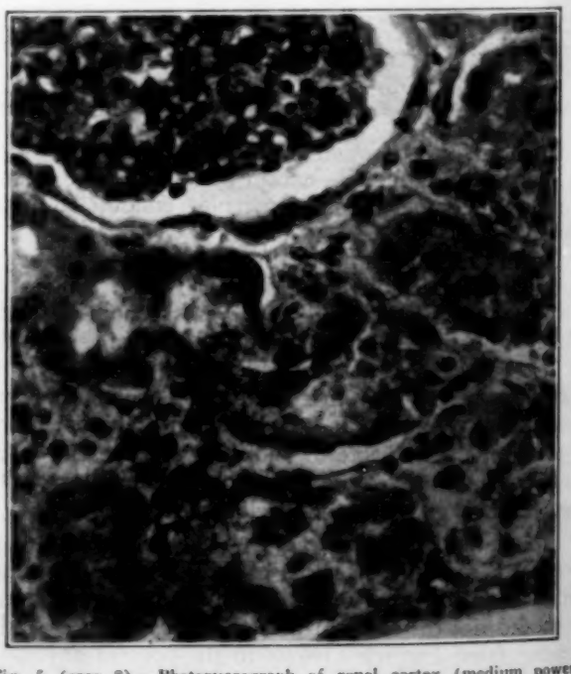

Fig. 5 (case 8).—Photomicrograph of renal cortex (medium power). The survival time was forty-six hours. Different degrees of necrosis are shown in the proximal convoluted tubules. Note the complete necrosis of the proximal convoluted tubules in contrast to the well-preserved nature of the malpighian corpuscle.

36. Tilp, cited by Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1925, vol. 6, pt. 1, pp. 583-584.

37. Helmholtz, H. F.: Hydropic Degeneration Produced by Sucrose, J. Pediat. 3: 144-150, 1933.

with Wilms, Marchand, Vogt and others that there is no acute glomerulitis due to burns. This point, however, argues neither for nor against a circulating burn toxin.

Acute Interstitial Nephritis.—In 34 of our dogs and in 1 of our human patients (case 17) an acute interstitial nephritis was observed. In an apparently similar case reported by Raysky (1910) the patient died three days after being burned. In no instance in our experience was acute interstitial nephritis seen before the third day, and in no case did it occur in the absence of secondary infection of the kidney. Classic descriptions of acute interstitial nephritis have been published by Councilman [38] (1897), Reichel [39] (1905), Marchand [40] (1913) and Aschoff [41] (1913). Our cases showed numerous foci of lymphocytic and plasma-cell infiltration in the lymph spaces and in the interstitial spaces immediately adjacent to the capsules of Bowman and the small blood vessels. The cortex was predominantly involved, but there were areas of infiltration also in the loose connective tissue about the larger vessels and beneath the pelvic mucosa. Seldom did the foci of infiltration surround the entire capsule of Bowman; when they did, the adjacent capsular epithelium became swollen and necrotic. In some such areas the glomerular loops were also contracted and degenerating. In general, in the immediate vicinity of small foci of infiltration the tubules were not altered in appearance, but in the more extensive foci of infiltration the tubules were replaced by infiltrating cells. The foci often followed the medullary rays through the cortex as prominent dark blue streaks. It is not known whether the lymphocytes come from the lymphoid structures in and about the kidney or from the blood. In the opinion of Fahr [42] (1925) they represent a response by the patient to the presence of a powerful bacterial exotoxin, such as those formed in diphtheria, scarlet fever and certain wound infections. The data in our cases suggest the presence of such an exotoxin associated with secondary infection of the burn areas. Fahr stated that kidneys showing such lymphocytic infiltration might return to normal with perhaps some scarring.

Fig. 8.—Photomicrograph of renal cortex of dog 3096 (medium power). The survival time was ten days. Note the lymphocytic infiltration about the parietal wall of Bowman's capsule.

38. Councilman, W. T.: Pathology of Acute Interstitial Nephritis, Am. J. M. Sc. **114**: 23-44, 1897.
39. Reichel, H.: Ueber Nephritis bei Scharlach, Ztschr. f. Heilk. **26**: 72-103, 1905.
40. Marchand, F.: Ueber die Herkunft der Lymphozyten und ihre Schicksale bei der Entzuendung, Verhandl. d. deutsch. path. Gesellsch. **16**: 5-80, 1913.
41. Aschoff, L.: Pathologische Anatomie, Jena, Gustav Fischer, 1923, pp. 448-450.

COMMENT

In summary, the most important pathologic lesions in the kidneys following extensive burns in the animal and human series here reported were found to be: (1) degenerative changes in the tubules; (2) congestion of the capillaries and venules; (3) tubular casts of the products of intravascular hemolysis, and (4) lesions due to infection.

The two principal pathologic–physiologic mechanisms which are believed to cause this damage are: shock and intravascular hemolysis. The significance of shock is twofold: Prolonged shock produces irreversible tubular degeneration and causes extrarenal uremia. With a fall of systolic blood pressure of 50 per cent, the volume of blood circulating through the kidney may be reduced 95 per cent, according to Richards [43] (1944). The result is oliguria or anuria with extrarenal azotemia.

The significance of intravascular hemolysis is less well understood. Earlier writers reported hematuria in a small minority of burn cases. Goodpastor (1946) found hemoglobinemia in all of his 20 patients with renal lesions and in all but 6 of the remaining 27 without pathologic changes in the kidneys. The frequency of intravascular hemolysis in the presence of extensive cutaneous burns is strikingly demonstrated by these figures.

Monke and Yuile [44] (1940) showed in experimental studies on dogs that the excretion of hemoglobin has a true renal threshold—about 100 mg. per hundred cubic centimeters—above which it is eliminated by the kidney at a rate directly proportional to its concentration in the plasma. They offered considerable evidence to show that, below this level, the hemoglobin in the glomerular filtrate is completely absorbed by the renal tubules, without injury. Recently Yuile, Gold, and Hinds [45] (1945) have reviewed the literature on the toxicity of the products of hemolysis on the kidney and, on the basis of their experimental material, have asserted that precipitation of hemoglobin in the renal tubules, and hence renal tubular necrosis, is primarily dependent on antecedent damage to the tubules and not on the acidity or alkalinity of the urine or the amount of hemoglobin in the blood. This antecedent damage to the kidneys may be produced by ischemia, shock or chemical poisoning. It is etiologically nonspecific.

Others have speculated on the possibility of a more toxic product of intravascular hemolysis, from either

42. Fahr, T., in Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1925, pp. 362-365.
43. Richards, D. W.: Circulation in Traumatic Shock in Man, Bull. New York Acad. Med. **20**: 377, 1944.
44. Monke, J. V., and Yuile, C. L.: The Renal Clearance of Hemoglobin in the Dog, J. Exper. Med. **72**: 149-165, 1940.
45. Yuile, C. L.; Gold, M. A., and Hinds, E. A.: Hemoglobin Precipitation in Renal Tubules, J. Exper. Med. **82**: 361-373, 1945.

the erythrocyte stroma or a derivative of hemoglobin, as the cause of the extensive tubular necrosis about the hemoglobin casts.

There is a striking similarity between the hematuric "burn" kidney and the renal observations in other conditions characterized by intravascular hemolysis, such as those following incompatible transfusions of blood, in hemolytic reactions to sulfonamide therapy and in malaria. The "crush syndrome" kidney, in which casts of a myohemoglobin are present, exhibits a comparable picture.

Finally, it may be stated that, while the renal lesions following extensive cutaneous burns are usually of a minor, secondary and probably reversible character, with little danger to the patient, there are in a minority of cases more serious lesions caused by prolonged shock or by a combination of shock and intravascular hemolysis, or by infection, which may lead to renal insufficiency, renal destruction and death.

REPORT OF CASES

Abstracts of the 20 human burn cases follow:

Case 1.—A white man aged 37 had second and third degree burns involving 90 per cent of the body surface received from steam in a railroad accident. The survival time was four to five hours; there were multiple fractures and a history of shock. Autopsy observations included: multiple pulmonary fat emboli, severe catarrhal bronchitis with ulceration, aspiration bronchitis, edema of meninges and brain substance and congestion and edema of the lungs. The renal examination showed gross cloudy swelling, mild congestion of the vessels including the glomeruli, severe albuminous degeneration and necrosis of the proximal convoluted tubules and thick ascending portion of Henle's loops, hyaline casts, submucosal hemorrhages in the kidney pelvis, moderately severe postmortem changes and mild degenerative fatty infiltration of the tubules.

Case 2.—A white girl aged 3 years fell into a brush fire. First, second and third degree burns involved over 50 per cent of the body surface. The survival time was seven hours. There was a history of severe shock. Observations at autopsy included: cerebral edema, aspiration of food into the bronchi, pulmonary congestion and edema, patchy emphysema and atelectases, with terminal right-sided cardiac dilatation. The kidneys showed congestion of vessels including glomeruli with red blood cells, decided albuminous degeneration and some necrosis of the proximal convoluted tubules and ascending thick portion of Henle's loop, with vacuolar degeneration of the collecting tubules in the cortex.

Case 3.—A white boy aged 5 had his clothes ignited while he was playing with matches. Second and third degree burns involved the entire trunk from pubis to chin including the neck and arms, with some charring. Sixty to 70 per cent of the body surface was involved. The survival time was twelve hours, with a history of severe shock and hematemesis. The treatment had been débridement and 5 per cent tannic acid spray. Autopsy observations included acute catarrhal bronchitis, pulmonary congestion and edema. The kidneys showed mild congestion of the blood vessels, including the glomeruli, with erythrocytes; there was albuminous degeneration and necrosis of the proximal convoluted tubules.

Case 4.—A Negro woman aged 50 had the anterior surface of the body, comprising 30 per cent of the body surface, burned with steam or hot water. She suffered first and second degree burns. The survival time was less than twenty-four hours; the exact time is not known. There was a history of shock. At autopsy the observations were: right-sided cardiac dilatation with relative tricuspid and pulmonary insufficiency; atrophy, passive congestion and parenchymatous degeneration of all organs; syphilitic aortitis, and lipoidosis of the adrenals. In the kidneys the gross observations were as follows: The capsule stripped with some increased difficulty; the surface was slightly granular. Acute passive congestion, atrophy and one old infarct were seen. Microscopic examination revealed congestion of the vessels and glomeruli. There were albuminous degeneration and necrosis of the proximal convoluted tubules and ascending thick portion of Henle's loops, interstitial edema in the medulla, hyaline casts, sclerosis of the vessels and postmortem changes.

Case 5.—A white woman aged 80, whose clothes ignited while she was smoking in bed, had 75 per cent of the body surface involved in second and third degree burns. The survival time was twenty-four hours. Treatment consisted in petrolatum gauze and pressure bandages, plasma and fluids for shock. There was a history of oliguria (60 cc. after burn). Autopsy examinations showed acute tracheobronchitis, pulmonary congestion and parenchymatous degeneration of the liver and adrenals. The kidneys on gross examination showed atrophy; the capsule stripped with slightly increased difficulty, leaving a finely granular surface; congestion was evident. Microscopic study revealed some congestion of the arterioles, capillaries and small veins but not of the glomeruli; also evident were glomerular endothelial hyperplasia, interstitial edema of the medulla, albuminous degeneration of the proximal convoluted tubules and thick ascending portions of Henle's loop and sclerosis of the vessels.

Case 6.—A white girl aged 3 years suffered second and third degree burns from the explosion of a gasoline stove, with 25 per cent of the body surface burned. The survival time was twenty-eight hours. A history of "toxic encephalitis" and terminal bilateral pneumonia was recorded. Autopsy observations were: acute passive congestion of all organs, petechial hemorrhages in the lungs, moderate parenchymatous degeneration of heart, liver and kidneys, acute catarrhal gastroenteritis and congestion and edema of the brain. The kidneys on gross examination showed severely congested, ground-glass appearance on cut section. Microscopic study disclosed congestion of the vessels and glomeruli with erythrocytes, albuminous degeneration of the tubules and ascending thick portion of Henle's loops, slight generalized glomerular endothelial hyperplasia, no lipoidosis and some foci of round cell infiltration.

Case 7.—A Mexican girl 2 years of age fell into a tub of scalding water. There were second and third degree burns over 35 per cent of the body surface. The survival time was forty hours. Tannic acid spray, 5 per cent, and blood transfusions were included in the treatment. There was a history of convulsions and pulmonary edema. At autopsy there was seen dehydration of all tissues with anhydremia, acute parenchymatous degeneration of the liver with beginning fatty metamorphosis, hemorrhage into the right adrenal gland and lymphadenopathy of the mesenteric glands. The kidneys on gross examination showed extreme congestion. Microscopic study disclosed congestion of the vessels with erythrocytes, albuminous degeneration of the proximal convoluted tubules and ascending thick portion of Henle's loops. There was some postmortem change.

Case 8.—A white man aged 38 was burned in an automobile accident. He had first, second and third degree burns involving 40 per cent of the body surface. The survival time was forty-six hours. He was treated with tannic acid and there was a history of shock and anuria. At autopsy the observations were: generalized edema, gangrene of the ileocecal region and sigmoid with thrombosis of the mesenteric veins and hemorrhage into the gastrointestinal tract, localized fibrinopurulent peritonitis, acute purulent bronchitis and lobar pneumonia, thrombi in the pulmonary arterioles, severe esophagitis and a small infected infarct of the adrenal. The gross renal examination showed some edema. Microscopic study of the kidneys revealed congestion of the vessels and glomeruli, petechial hemorrhages

beneath the pelvic mucosa and in the medulla, casts of erythrocytic débris in the collecting tubules, albuminous degeneration and simple necrosis of the epithelium of the proximal convoluted tubules and ascending thick portion of Henle's loops. Abundant protein débris was present in the tubules; there was a slight arteriolosclerotic nephropathy, with mild fatty infiltration of the collecting tubules.

CASE 9.—A white girl aged 6 years, whose clothes caught fire from a gas stove, had 45 per cent of the body surface involved in first, second and third degree burns. The survival time was forty-eight hours. Treatment included débridement, 7 per cent tannic acid jelly, adrenal cortical extract and blood transfusions. The history told of pulmonary edema and oliguria, prolonged and repeated emesis. There was a coroner's autopsy. Sections of heart, lungs, spleen, kidneys and liver were returned to Henry Ford Hospital. Autopsy observations were: edema and congestion of the lungs and myocardial degeneration. The kidneys exhibited mild congestion of the vessels but not of glomeruli, albuminous degeneration and simple necrosis of the proximal convoluted tubules and the ascending thick portion of Henle's loop. There were casts of erythrocytic débris in the collecting tubules.

CASE 10.—A white girl aged 2½ years sat in a bucket of hot water. She suffered first and second degree burns which involved 50 per cent of the body surface. The survival time was seventy-five hours. The burns were treated with tannic acid jelly and the child received repeated transfusions of blood. There was a history of prolonged stupor, acidosis and rapidly progressive anemia. Autopsy observations included: multiple petechial hemorrhages in the subperitoneal connective tissue, mesentery, omentum and gastrointestinal tract and beneath the visceral pleura; there was hemorrhage into the gastrointestinal tract; generalized edema was present with 400 cc. of ascitic fluid and 100 cc. of pleural fluid in each pleural cavity; decided parenchymatous degeneration of the liver had taken place; pulmonary congestion and hemorrhage were evident. The kidneys on gross examination exhibited several small hemorrhages in the peripelvic tissue and along the left ureter. The microscopic study showed widespread severe hydropic degeneration of the proximal convoluted tubules. ("Patient expired shortly after receiving 100 cc. of 25 per cent glucose" and 100 cc. of blood.) There was mild congestion of the vessels but not of the glomeruli. Erythrocytic débris was seen in the collecting tubules.

CASE 11.—A white boy aged 2½ years died two days after extensive second degree burns involving the abdomen, axilla, back, buttocks, genitalia and lower extremities. There was a history of pulmonary edema and symptoms of toxicity. The autopsy showed: acute purulent bronchitis and fibrinopurulent lobar pneumonia; severe pulmonary congestion; edema and hemorrhage; patchy emphysema and atelectasis; lipoidosis of the myocardium, liver, adrenal cortex and kidneys, and intense acute passive congestion and edema of all viscera. The kidneys showed gross evidence of congestion and cloudy swelling. The microscopic observations were: congestion of vessels and glomeruli, albuminous degeneration and simple necrosis of the proximal convoluted tubules and ascending thick portion of Henle's loop, mild glomerular endothelial hyperplasia and severe degenerative fatty infiltration of the proximal convoluted tubules.

CASE 12.—A Negro man aged 36 was burned by spilled molten iron, which ignited his clothes. Second and third degree burns involved 40 per cent of the body surface. The survival time was four days. Treatment consisted in the application of 7 per cent tannic acid jelly and the transfusion of blood. There was a history of bleeding from the gums the day before death. After the coroner's autopsy portions of liver, spleen, kidneys, adrenals and skin were returned to Henry Ford Hospital for sections. The autopsy showed generalized edema, ascites and hydrothorax, myocardial degeneration and extensive passive congestion of the liver, spleen and kidneys. Microscopic study of the kidneys showed congestion of the vessels but not of the glomeruli, albuminous degeneration of the proximal convoluted tubules and ascending thick portions of Henle's loops, necrosis of Bowman's capsule with intracapsular débris and granular casts in the collecting tubules.

CASE 13.—A white man aged 24 died four and a half days following first, second and third degree burns involving approximately 30 per cent of the body surface. Autopsy revealed moderate infection and early healing of burned areas; agglutination thrombi in the pulmonary capillaries; cerebral and meningeal edema; a pronounced degree of acute passive congestion of the liver and spleen; patchy interstitial myocarditis; acute catarrhal bronchitis; localized superficial ulceration of the larynx; patchy Zenker's necrosis of the diaphragm, and lymphocytic infiltration of the adrenal gland. The kidneys on gross examination revealed cloudy swelling. The microscopic study disclosed: congestion of the vessels and the glomeruli; albuminous degeneration and simple necrosis of the proximal convoluted tubules, and ascending thick portions of the loop of Henle; recent hemorrhage into the submucosa of the pelvis; no lipoidosis; necrotizing ulceration of the pelvic mucosa, and a basophilic precipitate in some collecting tubules (probably a sulfonamide compound). There was minimal glomerular endothelial hyperplasia.

CASE 14.—A white man aged 44 suffered second and third degree burns of the face, neck, hands, feet and right leg when a kerosene can exploded as he was lighting the kitchen stove. A history of secondary infection of burns and septicopyemia was recorded. The survival time was eight days. Autopsy revealed: pyemic abscesses of the lungs, myocardium, kidneys, testes and small intestine; acute fibrinopurulent pericarditis and endocarditis; acute purulent bronchitis and bronchopneumonia; pulmonary congestion, hemorrhage and edema; terminal cardiac dilatation with relative tricuspid and pulmonary insufficiency; acute passive congestion and parenchymatous degeneration of all organs. On gross examination of the kidneys several irregular light-colored areas were seen on the surface. Cut section showed these to be pyramidal, with the base at the periphery. Microscopic study revealed "multiple small embolic abscesses"; many of these contained colonies of cocci. There was albuminous degeneration of the proximal convoluted tubules and ascending thick portion of Henle's loop and congestion of the vessels and glomeruli. Lipoidosis was not observed.

CASE 15.—A white woman aged 16 received second and third degree burns from an open furnace door ten days before her death. Autopsy disclosed intense acute passive congestion of the lungs, moderate chronic pulmonary edema and a small hemorrhagic infarct in the lung, with central abscess. Also there was evidence of acute purulent bronchitis, petechial hemorrhages in the submucosa of the bronchioles and congestion of all viscera. The kidneys on microscopic examination exhibited congestion of the vessels and glomeruli, albuminous degeneration and simple necrosis of the proximal convoluted tubules and ascending limb of Henle's loops and minimal glomerular endothelial hyperplasia. There was no lipoidosis.

CASE 16.—A white boy aged 18 had his clothes catch fire while he was smoking in bed. There were second and third degree burns of the face, neck, thorax, right arm, abdomen, back, thighs and genitalia. From 60 to 65 per cent of the body surface was involved. The survival time was fourteen days. Autopsy disclosed acute purulent bronchitis and lobar pneumonia, older organizing fibrin in many alveoli and bronchi, bronchiolitis obliterans, pulmonary congestion and extensive hemorrhage, adhesive pleuritis, organizing mural thrombus in the heart, slight degenerative fatty infiltration of the myocardium, edema of the brain and meninges, widespread petechial hemorrhages beneath the serosal surfaces, hemorrhage and necrosis of the urinary bladder and colonic mucosa and submucosa, bone marrow giant cell emboli in the heart and renal glomeruli, shallow acute laryngeal ulcer, small fresh hemorrhagic splenic infarct and hydropericardium. Gross examination of the kidney revealed congestion. Microscopic study showed congestion of the vessels and glomeruli, albuminous degeneration and necrosis of the proximal convoluted tubules with calcification,

vacuolar degeneration of Henle's loops and the collecting tubules and small hemorrhages in the medulla. There was no lipoidosis.

CASE 17.—A white girl aged 8 years had her clothes catch fire while she was playing with matches. Death resulted fourteen days after extensive first and second degree burns of the neck, thorax, abdomen, back and arms. The treatment included tannic acid dressings. Autopsy revealed cerebral congestion and edema, subepicardial and pulmonary petechial hemorrhages, acute pulmonary edema, patchy atelectasis, degenerative fatty infiltration of the myocardium, liver and kidneys, slight chronic catarrhal tracheobronchitis and shallow acute esophageal ulcers. On gross examination the kidney was softer than usual, with cloudy swelling. Microscopic study revealed congestion of the vessels and glomeruli, lymphocytic infiltration beneath the pelvic mucosa, well advanced fatty degeneration of the tubular epithelium, moderate glomerular endothelial proliferation and acute interstitial nephritis with lymphocytic infiltration about blood vessels.

CASE 18.—A white boy aged 3 years died the twenty-fifth day after scalding water caused extensive superficial burns (first, second and third degree) involving the skin of the back, abdomen, thighs, arms and genitalia. There was a history of infection of the burned areas. Autopsy examination disclosed infected burns, pronounced acute passive congestion and parenchymatous degeneration of all viscera, edema of the left foot and the scrotum and emphysema. On cut section of the kidneys a triangular abscess was seen in the upper pole of the left kidney. Microscopic study revealed: albuminous degeneration of the proximal convoluted tubules and ascending thick portion of Henle's loops; congestion of the vessels; areas of recent hemorrhage; early embolic purulent nephritis with several small necrotic foci around small vessels containing infected emboli, and slight degenerative fatty infiltration of the tubular epithelium.

CASE 19.—A white boy 10 years of age was splashed with burning oil and 25 per cent of the body surface was involved in second and third degree burns. The survival time was twenty-six days; the burns were treated with tannic acid. Autopsy revealed: acute toxic encephalopathy; severe infection of unhealed burns with pyoderma; acute passive congestion and parenchymatous degeneration of the liver and kidneys; acute ulcerative and purulent esophagitis; agglutination of the red and white cells in the blood stream with multiple small emboli in the meninges and substance of the brain. The kidneys on gross examination were flabby, with cloudy swelling; they were moderately congested and minute yellow granules were adhering to the pelvic mucosa. Microscopic study disclosed congestion of the vessels, albuminous degeneration and simple necrosis of the proximal convoluted tubules, slight degenerative fatty infiltration of the tubules, minimal glomerular endothelial hyperplasia and sulfanilamide casts in the collecting tubules.

CASE 20.—A white man aged 38 had gasoline burns of second and third degree, which involved 70 per cent of the body surface. The survival time was twenty-nine days. The burns were secondarily infected. Observations at autopsy were as follows: infected burns; acute chronic adhesive pericarditis; vegetations of the aortic, pulmonary and tricuspid valves; subepicardial fatty infiltration; subendocardial degeneration and fatty infiltration; endocardial sclerosis with fibrosis extending into the myocardium; terminal right-sided and left-sided cardiac dilatation; chronic adhesive and acute fibrinous pleuritis; early acute purulent bronchitis; pulmonary congestion, edema and atelectasis; multiple infarctions in the spleen and kidneys, and passive congestion and parenchymatous degeneration of all the organs. The kidneys on gross examination exhibited a smooth surface with mottled yellow areas in purple-red background; yellow areas were noted in the cortex on cut section. Microscopic study showed multiple infected infarcts, metastatic abscesses, congestion of the tubules and thick ascending portions of the loops of Henle and minimal glomerular endothelial proliferation.

CRUSH SYNDROME: POST-TRAUMATIC ANURIA

Observations on Genesis and Treatment

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Traumatic shock is thought of as an acute state from which no threatening residues persist. As regards recovery of renal function, doubt was cast on this view by reports from the scene of the Messina earthquake and by detailed studies of cases of post-traumatic uremia occurring in the German Army in World War I.[1] Crush syndrome was rediscovered, named and studied on a wider scale during the London blitz.[2] Similar cases have been reported from the accidents of civilian life.[3] Many others, civil and military, have probably gone undiagnosed because fatal shock and sepsis have probably overshadowed the defect of renal function.

Typically, the syndrome is initiated by crush of a limb or occlusion of its arterial supply for several hours. Release from débris or restoration of blood flow is followed by swelling, with whealing at the sites of compression. The limb becomes hard, paralyzed and anesthetic. Loss of plasma into the area of injury causes hemoconcentration. Shock may supervene and be relieved by transfusion. Urine passed during the first twenty-four hours is colored red or brown with myoglobin, contains albumin and shows many brown and black granular pigment casts. Urinary output and specific gravity thereafter diminish. The patient passes into restless delirium with thirst and vomiting. Arterial pressure and concentration of serum potassium are increased. Azotemia rises to about the sixth day, when death suddenly occurs in about two thirds of the cases. The volume of urine of those who are to recover increases about this time, and their renal function is slowly restored in succeeding months.

The anuria which may follow intravascular hemolysis, as by transfusion of incompatible blood, is familiar. Myoglobin, the red pigment of muscle, has the same basic structure as hemoglobin. The fact that its molecule is only a fourth as large permits it to be filtered through glomerular capillaries into renal tubules at about twenty-five times the rate of hemoglobin. Renal failure in crush syndrome was then, by analogy, attributed to deposition of myoglobin casts in the renal tubules.

The exact mechanism of hemoglobinuric nephrosis is not established. There is compelling evidence which associates it with urinary acidity. Renal damage in crush syndrome was therefore attributed to the coincidence of myoglobinuria—due to release of myoglobin from crushed muscle—and aciduria—due to the outpouring of lactic and phosphoric acids from damaged muscle cells.

From the Research Division of the Cleveland Clinic Foundation.
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3. McClelland, J. C.: Anuria: A Report of Three Types of Cases, Canad. M. A. J. **45**: 332, 1941. Husfeldt, E., and Bjering, T.: Renal Disturbances in Traumatic Shock, Hospitalstid. **78**: 781, 1935. Caplan, A., and Dunkerley, G. E.: Traumatic Anuria in Miner, Lancet **1**: 147, 1945.

Others thought it more likely that renal injury in crush syndrome is a consequence of prolonged arterial hypotension with renal ischemia and anoxia; they viewed the blocking of renal tubules with pigment as more a result than a cause of renal damage.

Another view attributes renal injury to toxic substances liberated from injured tissues, either acting directly on renal tubule cells or acting indirectly by decreasing the rate of renal blood flow.

These differences in the point of view are significant, for prevention and treatment of crush syndrome depends on a knowledge of the mechanism which must be countered.

MECHANISM AND TREATMENT OF CRUSH SYNDROME

I. *The Pigment Theory.*—Our experience on the effect of intravenous injection of myoglobin and hemoglobin in normal dogs [4] and in rats with acid urine shows that the renal injury thus produced is highly variable and seldom serious. The renal function most depressed is that of tubular secretion. The degree of impairment is directly proportional to the amount of pigment retained in the kidneys. The amount of pigment retained does not depend directly on urinary p_H within the range 5.0 to 5.8. Rather, renal injury and pigmentary retention seem to depend on the coincidence of aciduria with oliguria (see the accompanying table).

Others [5] have observed renal injury which is occasionally severe in rabbits with acid urine injected with human myoglobin. Our conclusions vary from theirs in that we were unable to cause fatal uremia in normal animals and in our attribution of renal damage to the combination of aciduria with oliguria.

The fact that oliguria as well as aciduria is a requisite of myoglobin renal damage seems to explain some of the divergent reports on the renal effects of hemoglobinuria.[6] Other mechanisms of hemoglobinuric injury have been invoked, as in the suggestion that injection of hemoglobin solutions causes severe renal vasoconstriction.[7] This is not borne out in our experience, for injections of sterile solutions of both myoglobin and hemoglobin and even the initiation of intravascular hemolysis and hemoglobinuria cause no change in renal blood flow. The discrepancy may result from the injection of nonsterile solutions of hemoglobin.

Hemoglobinuric and myoglobinuric renal damage seem largely dependent on the precipitation of pigment casts within the renal tubules. This is most likely to occur when the urine is highly concentrated, i. e., oliguric and acid. These casts injure by causing hydronephrosis of nephrons and groups of nephrons. Pressure from distended nephrons may interfere with the function of some which are still patent. It is not clear that this is the sole mechanism of renal injury from pigment precipitation. Hematin is cytotoxic and when intravenously injected in large amounts causes severe renal as well as hepatic injury.[8] Myoglobin and hemoglobin in acid solution tend to form metamyoglobin and methemoglobin, respectively, and to be precipitated as such. Hematin is easily liberated from these metaforms of the pigments by tissue and urinary enzymes. Part of the renal tubular damage in hemoglobinuria and myoglobinuria may be due to release of hematin from precipitated heme-globin pigments.

II. *Shock, Hypotension and Renal Ischemia.*—Clamping the renal artery for periods of more than an hour results in severe renal structural and functional damage [9] with prolonged ischemia, degeneration largely of the proximal convoluted tubules, azotemia and oliguria, loss of the secretory aspects of renal function and impairment even of the normal selective impermeability of renal tubules. This last injury creates a glomerulovenous circulation of tubular fluid which tends to increase oliguria due to ischemia and may facilitate pigmentary aggregation in the tubules. But clamping of the renal artery reduces renal blood flow to less than 10 per cent of normal. It is not at first apparent that this low level of blood flow can be present in clinically recoverable shock and in the absence of severe hypotension.

Pertinent to this problem, we have shown that successive periods of prolonged hypotension and early shock due to bleeding result in progressive renal vasoconstriction, such that restoration of arterial pressure and blood volume to normal by transfusion fails to restore renal blood flow to more than 50 per cent of normal.[10] To mimic more closely the conditions of crush syndrome we turned next to studies of renal blood flow and function in dogs to which tourniquets had been applied [11] which all but obliterated arterial inflow to the limbs. Within a few minutes after beginning the experiments, renal blood flow was reduced to 50 per cent of normal, and at the end of an hour it was down to about 20 per cent of normal. Interruption of vasomotor pathways by renal denervation, prevention of hemoconcentration and transfusion with restoration of arterial pressure and blood volume are measures which restore only a fraction of the normal renal blood flow. Concurrent measurements of the vasoconstrictor properties of plasma show that a vasoconstrictor substance appeared in it with the onset of

Effects on Renal Function of Myoglobin Injection in Rats

Group		Blood Urea Nitrogen, Mg./100 Cc. 24 Hrs. After Injection	48 Hrs. After Injection	72 Hrs. After Injection	No. of Animals in Group
1	Aciduria (p_H 5.2).....	17	17.5	..	16
2	Aciduria (p_H 5.0).....	32	29.0	20	12
3	Aciduria (p_H 5.1) with prior dehydration	58	36.0	28	15

Effect of aciduria alone at two levels of urinary p_H and of aciduria with prior deprivation of water for twenty-four hours in normal rats given 40 mg. of beef metamyoglobin intravenously. Values given for urinary p_H and blood urea nitrogen are means of the observations in each group.

4. Corcoran, A. C., and Page, I. H.: Renal Damage from Ferroheme Pigments Myoglobin, Hemoglobin, Hematin, Texas Rep. Biol. & Med. **3**: 528, 1945.

5. Bywaters, E. G. L., and Stead, J. K.: The Production of Renal Failure Following Injection of Solutions Containing Myohemoglobin, Quart. J. Exper. Physiol. **33**: 53, 1944.

6. Foy, H.; Altmann, A.; Barnes, H. D., and Kondi, A.: Anuria, with Special Reference to Renal Failure in Blackwater Fever, Incompatible Transfusions and Crush Injuries, Tr. Roy. Soc. Trop. Med. & Hyg. **36**: 197, 1943. Ross, J. F.: Hemoglobinemia and the Hemoglobinurias, New England J. Med. **233**: 691, 732 and 766, 1945.

7. Hesse, E., and Filatov, A.: Experimentelle Untersuchungen über das Wesen des hämolytischen Schocks bei der Bluttransfusion und die therapeutische Beeinflussung desselben: die Nierenfunktionsstörungen im akuten Experiment, Ztschr. f. d. ges. exper. Med. **86**: 211, 1933. Mason, J. B., and Mann, F. C.: Effect of Hemoglobin on Volume of Kidney, Am. J. Physiol. **98**: 181, 1931.

8. Anderson, W. A. D.; Morrison, D. B., and Williams, E. F., Jr.: Pathologic Changes Following Injections of Ferrihemate (Hermatin) in Dogs, Arch. Path. **33**: 589 (May) 1942. Corcoran and Page.[4]

9. Scarff, R. W., and Keele, C. A.: Effects of Temporary Occlusion of Renal Circulation in Rabbits, Brit. J. Exper. Path. **24**: 147, 1943. Phillips, R. D., and Van Slyke, P. D.: Personal communication to the authors. Selkurt, E. E.: Renal Blood Flow and Renal Clearance During Hemorrhagic Shock, Am. J. Physiol. **145**: 699, 1946.

10. Corcoran, A. C., and Page, I. H.: Effects of Hypotension Due to Hemorrhage and of Blood Transfusion on Renal Function in Dogs, J. Exper. Med. **78**: 205, 1943.

11. Corcoran, A. C.; Taylor, R. D., and Page, I. H.: Immediate Effects on Renal Function of the Onset of Shock Due to Partially Occluding Limb Tourniquets, Ann. Surg. **118**: 871, 1943.

renal ischemia. Such a substance also appears in animals subjected to bleeding or burns.[12] The demonstration of a vasoconstrictor in the plasma suggests that the vasoconstrictive ischemia is of humoral origin. A similar renal ischemia occurs in shock in human beings.[13]

Since even the early stage of shock, and more particularly of shock elicited by injury to skeletal muscles, is associated with an intense renal vasoconstrictive ischemia, it seems logical to attribute the phenomena of crush syndrome to persistent renal ischemia with resultant tubular renal injury.[14]

Unfortunately, in the dog, shock severe enough to result in intense renal ischemia is almost always fatal, so that nothing corresponding to crush syndrome was observed in a large series of experiments. Further, the muscles of dogs contain myoglobin, so that the question whether crush syndrome results from renal ischemia alone or from the coincidence of renal ischemia and myoglobinuria could not be answered. We therefore turned to studies in rats. The muscles of these animals

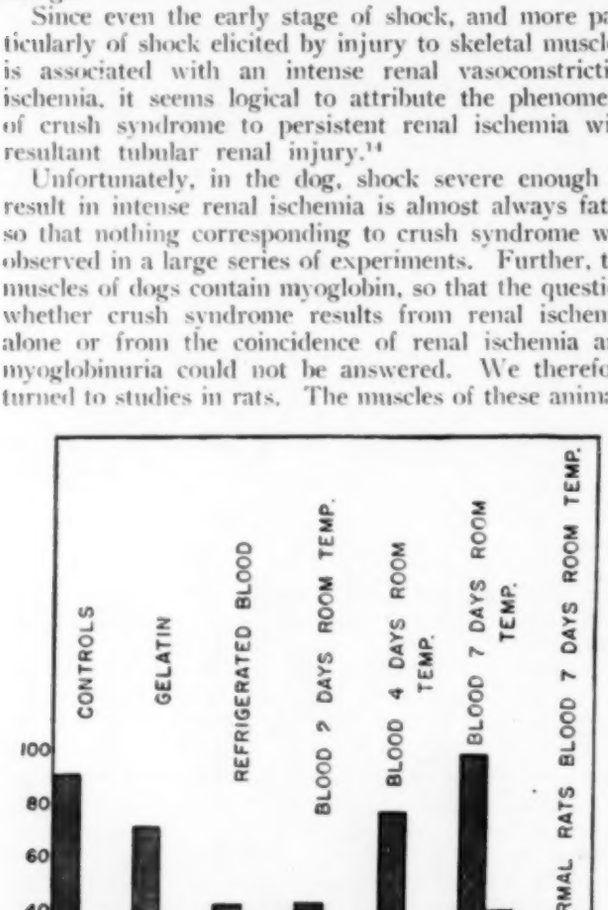

Chart 1.—Comparative levels of blood urea nitrogen and mortality (1) in normal rats given deteriorated blood and (2) in crushed, myoglobinuric rats treated with intravenous injection of gelatin and of blood under various conditions of preservation. The blood urea nitrogen level (mg./100 cc., in the crosshatched columns) is the mean of observations made forty-eight hours after treatment. The mortality (diagonally shaded columns) is given as the per cent of animals dying within seventy-two hours of treatment.

do not contain much myoglobin. Their resistance to shock seems to be greater than that of dogs.

Two groups of rats were subjected to crushing injury of one hind leg. One group received myoglobin solution intravenously one hour after release of the crush and the other, an equal volume of isotonic solution of sodium chloride.[15] Moderate azotemia was frequent in rats not given myoglobin and was accompanied by minor renal cytologic changes, notably cloudy swelling and the formation of hyaline casts. In contrast, severe and frequently fatal renal failure, accompanied with severe degenerative changes in the distal convoluted tubules and accumulation therein of pigment casts occurred in the group crushed and injected with myoglobin.[16]

We concluded that renal failure in crush syndrome is due to the coincidence of renal vasoconstrictive ischemia with myoglobinuria.

The mechanism of crush syndrome can thus be visualized. Vasoconstrictive renal ischemia occurs with the onset of shock or even of injury. The urine is acid and small in volume. The muscle cells are fragmented, so that when circulation is restored through crushed areas myoglobin diffuses into the blood, whence it is filtered into the renal tubules. Thus the conditions of pigmentary precipitation (aciduria, oliguria) are met, while the kidney is further damaged by lack of blood flow. It has been demonstrated[17] that hemoglobin precipitation in renal tubules is facilitated by prior injury, whether from ischemia or injection of a nephrotoxin (tartrate). However, it is not clear from these experiments whether the factor responsible was cellular injury as such or oliguria resulting therefrom. In either case the conditions of precipitation are present in crush syndrome.

III. *Hemoglobinuria.*—The participation of myoglobinuria in crush syndrome raises the question of hemoglobinuria and of its effect. This problem was explored in many of the early cases of crush syndrome, in which it was definitely shown that the patients' urinary pigment was myoglobin and not hemoglobin.[18] But 8 cases of renal failure have been reported following war injury,[19] in at least 2 of which the renal injury of the patients seems to have resulted from intravascular hemolysis following transfusion of compatible blood.

Gibson, Aub and Evans[20] and their co-workers have shown that refrigerated whole blood preserved in acid citrate medium can be transfused with complete safety (less than 10 to 20 per cent hemolysis of donor cells in four hours after transfusion). Short periods of exposure to room temperature greatly shortens the life of the transfused cells. Several days of such exposure need not result in visible hemolysis of the blood in the flask, but nearly all the cells of such blood may break down in less than four hours in the recipient's circulation, with resultant hemoglobinemia.

In collaboration with these workers we therefore examined the safety, as regards renal function, of blood stored under various conditions when transfused in normal rats with neutral and acid urine and in rats with one crushed limb. In some experiments the survival of donor cells was traced by tagging their hemoglobin with radioactive iron (Fe^{59}).

Results of these experiments are summarized in chart 1. Transfusion of sterile, refrigerated blood which has been preserved for one or two weeks greatly decreases the amount of renal injury which results from crushing and myoglobin injection in untreated rats. Exposure of such blood to room temperature for two days does not impair the benefit of the trans-

12. The nature of the vasoconstrictor is unknown. It is not angiotonin or angiotonin-like, neither is it adenosine or histamine. Page, I. H.: Occurrence of a Vasoconstrictor Substance in Blood During Shock Induced by Trauma, Hemorrhage and Burns, Am. J. Physiol. **130**: 386, 1943.

13. Lauson, H. D.; Bradley, S. E., and Cournand, A.: Renal Circulation in Shock, J. Clin. Investigation **23**: 381, 1944.

14. Maegraith, B. G.; Havard, R. E., and Parsons, D. S.: Renal Syndrome of Wide Syndrome Distribution Induced Possibly by Renal Anoxia, Lancet **2**: 293, 1945.

15. Corcoran, A. C., and Page, I. H.: Post-Traumatic Renal Injury: Summary of Experimental Observations, Arch. Surg. **51**: 93 (Sept.) 1945.

16. Dr. Paul N. Harris, Eli Lilly and Company Research Laboratories, kindly undertook the appraisal of structural changes in experimental crush syndrome. Drs. Tracy Mallory and Balduin Lucké confirmed the opinion that the changes produced in crushed, myoglobinuric rats closely mimicked those of crush syndrome.

17. Yuile, C. L.; Gold, M. A., and Hinds, E. G.: Hemoglobin Precipitation in Renal Tubules, J. Exper. Med. **82**: 361, 1945.

18. Bywaters, E. G. L.; Delorg, G. E.; Rimington, C., and Smiles, J.: Myohaemoglobin in the Urine of Air Raid Casualties with Crushing Injury, Biochem. J. **35**: 1164, 1941.

19. Darmody, E. M., and others: Traumatic Uraemia: Reports on Eight Cases, Lancet **2**: 809, 1944.

20. Gibson, J. G., 2d; Evans, R. D., and Aub, J. C.: Personal communication to the authors.

fusion, unless the blood is contaminated by bacteria. Infected blood was almost instantly lethal in crushed rats.

The benefit due to transfusion in experimental crush syndrome is lost when the blood has been maintained at room temperature for four days, and blood kept at room temperature for seven or nine days is definitely injurious to crushed rats. The injury such blood causes is largely renal. Calculations made by Dr. Gibson show that in the rat, as in the human being, such blood is rapidly hemolyzed (chart 2) in the recipient's circulation. About 40 per cent of the hemoglobin present in the transfusion (50 per cent of that hemolyzed in the circulation) appears in the kidneys.

In strong contrast, transfusion of blood maintained at room temperature for more than a week under sterile conditions into normal rats did not cause either functional or anatomic renal injury.

It is therefore apparent, as the paper of Darmody and others [19] has suggested, that what seems to be crush syndrome may result from intravascular hemolysis. Blood not visibly hemolyzed but inadequately refrigerated may be lethal in injured subjects, although harmless enough in normal recipients.

IV. *Renal Toxins in Crush Syndrome: Allied States.*—The major renal lesion in crush syndrome is epithelial. Renal epithelium is sensitive to a variety of toxins. Thus, a cellularly toxic substance liberated into the blood during states of shock and injury might participate in the renal damage caused by renal ischemia and the precipitation of pigment. Most toxins injure the proximal tubule more than they do the distal. This hypothetic toxin would have to differ from known nephrotoxins in that it would act in the distal convoluted tubules, for it is in these that the renal injury of crush syndrome is concentrated. No such directly-acting cellular toxin has been demonstrated.

There may be a variety of vasoactive substances in the blood during shock and states of severe injury which tend to damage renal epithelium indirectly by depressing blood flow. The vasoconstrictor substance of shock [12] has been previously noted. Eggleton [21] has shown that from injured muscle something is released into the blood and partially destroyed in the liver and that this substance depresses renal function, presumably by decreasing renal blood flow. Bielschowsky and Green [22] suggested that adenosin pyrophosphate and similar substances liberated from muscle might account for some of the vascular collapse, including that of the kidneys. Bollman and Flock [23] have since shown that adenosine triphosphate cannot account for the effects of tourniquet shock. A vasoexcitor substance of supposed renal origin has been found in plasma in early shock.[24] But it would be inaccurate to refer to these as renal toxins, since their effect on renal function seems to be indirect and due to the renal ischemia they may provoke.

The case for the primacy of renal ischemia is not complete. The principal renal lesion which results from complete obliteration of renal arterial inflow is degeneration of the proximal convoluted tubules, which resembles that of cortical renal necrosis. The localization of renal damage in crush syndrome in distal tubules suggests that ischemia of crush syndrome must differ from that which follows clamping of the renal artery. We [10] have shown that renal blood flow is irregularly distributed during states of hypotension, and it may be that this irregularity of distribution accounts for the distal tubular localization.[24a]

Whatever the mechanism by which the injury concentrates in this site, it is increasingly evident that such damage occurs in a wide variety of conditions. In many of these the major cause seems to be liberation of heme-globin pigments into the blood (transfusion-induced hemolysis, burns, blackwater fever, other hemoglobinurias, crush syndrome and other myoglobinurias). But there are many states in which liberation of pigment into the blood is not a major concomitant (yellow fever, spirochetal jaundice, Asiatic cholera, typhus fever, postpartum shock, shock due to obstetric accidental hemorrhage, and that due to exposure to high altitudes). In these also renal failure

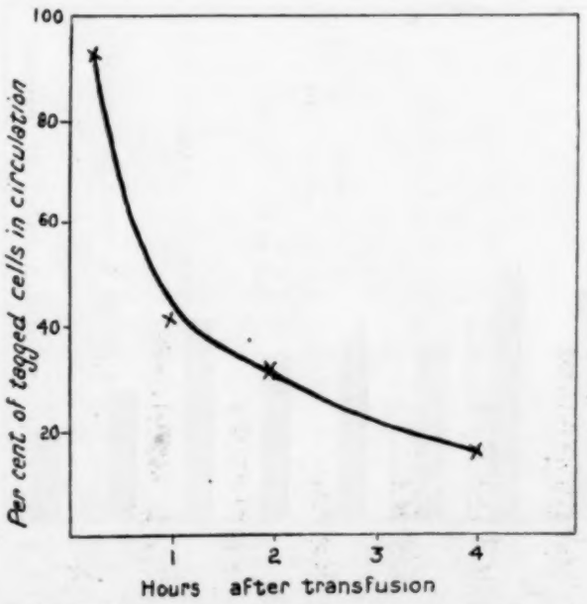

Chart 2.—Course of destruction in the recipient's circulation of transfused blood kept at room temperature for seven to nine days. (Data provided by Dr. John G. Gibson 2d.)

may occur with distal tubular injury and accumulation in the tubules of variable numbers of casts, including some composed of pigment. Similar injury, usually without pigmentary cast accumulation, occurs in the anuria of delayed hepatorenal syndrome and in that of diabetic coma.

In most of these conditions there is widespread damage of tissue, and it may be assumed that vasoconstrictive renal ischemia is present. The pigment casts may form from intravascularly liberated hemoglobin or may form intratubularly from small glomerular capillary leak of red cells. The primary lesion is ischemia, and tubular obstruction and injury from the formation of casts is secondary and additive. Peters [25] has suggested that increased intrarenal pressure due to tubular obstruction and inflammatory edema is a further mechanism of renal damage in these disease states, although the evidence for this is incomplete. Thus, the mechanisms of what has been recently termed "lower nephron nephrosis" are probably multiple.

21. Eggleton, M. G.: Crush Kidney Syndrome in the Cat, Lancet **2**: 208, 1944.
22. Bielschowsky, M., and Green, H. N.: Shock-Producing Factors from Striated Muscle: Fractionation, Chemical Properties and Effective Doses, Lancet **2**: 153, 1943.
23. Bollman, J. L., and Flock, E. V.: Changes in the Phosphate of Muscle During Tourniquet Shock, Am. J. Physiol. **142**: 290, 1944.
24. Shorr, E.; Zweifach, B. W., and Furchgott, R. F.: On the Occurrence, Sites and Modes of Origin and Destruction: Principles Affecting the Compensatory Vascular Mechanisms in Experimental Shock, Science **102**: 489, 1945.

24a. See the addendum.
25. Peters, J. T.: Oliguria and Anuria Due to Increased Intrarenal Pressure, Ann. Int. Med. **23**: 221, 1945.

V. *Treatment.*—Treatment is directed toward the underlying mechanisms. These mechanisms in crush syndrome with myoglobinuria or hemoglobinuria are, in their probable order of significance, (*a*) vasoconstrictive renal ischemia; (*b*) myoglobinemia and myoglobinuria or hemoglobinemia and hemoglobinuria with (*c*) oliguria and (*d*) aciduria and (*e*) tubular cellular injury due to renal ischemia. In the allied states of anuria associated with distal tubular lesions but without pigmentary participation the factors responsible are apparently (*a*) renal ischemia and (*b*) resultant cellular injury.

The primary problem in both types of renal damage is renal ischemia. This renal ischemia is due to vasoconstriction. The vasoconstriction results largely from the action of a vasoconstrictor present in blood, which in our view is liberated from injured tissues. Thus the primary issue is to decrease the supply of vasoconstrictor by (1) slowing its absorption and (2) decreasing its formation.[26] The absorption of such a substance from injured tissue is slowed and the renal

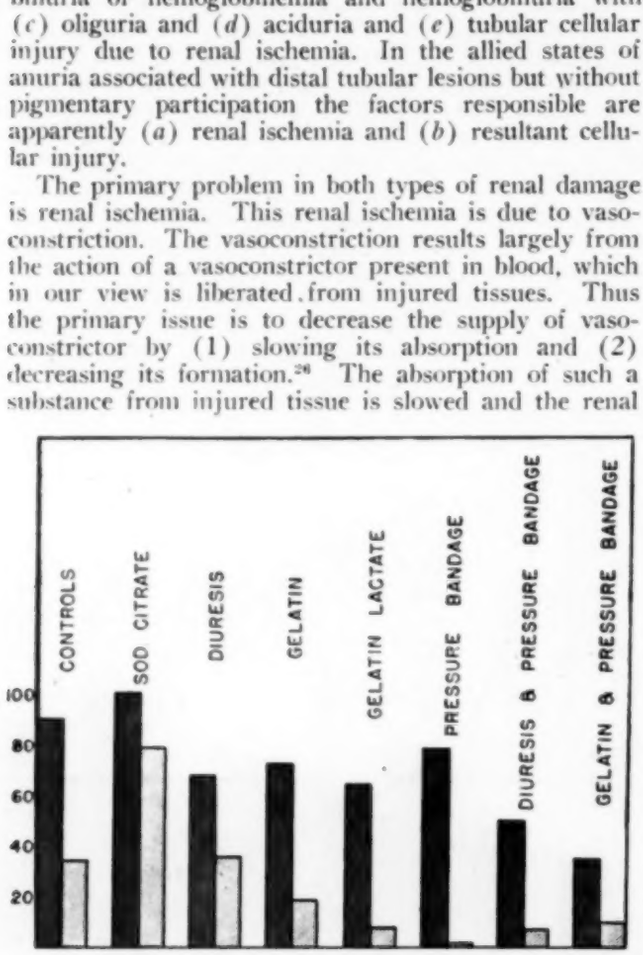

Chart 3.—Effect of various forms of treatment on experimental crush syndrome. Data are summarized as in chart 1.

ischemia minimized by use of a compression bandage.[11] The formation of vasoconstrictor seems to be due in part to circulatory failure; consequently, it should be slowed by restoration of circulatory function, i. e., by transfusion and similar measures. It may be that refrigeration would decrease both formation and liberation of vasoconstrictor.

Oliguria in these conditions is due in part to renal ischemia with decreased glomerular filtration and, it would seem, also to excessive reabsorption of water through injured tubular cells. It is countered by the restoration of blood volume and blood pressure, by decreasing renal ischemia and by the use of diuretics.

Aciduria in crush syndrome is due to absorption of acid metabolites from injured muscle. This absorption is slowed by application of pressure bandages. Urinary acidity may then be decreased by the supply of alkali, oral or intravenous, as sodium lactate, bicarbonate or citrate.

Finally, if there exists a nephrotoxin acting on the tubular cells from the tubular fluid, it is apparent that restoration of a rapid rate of urinary flow will decrease its local concentration.

Thus, the basic preventative measures in crush syndrome are (1) pressure bandaging of the injured limb, (2) rapid treatment of shock with restoration of blood volume and blood pressure, (3) provision of adequate fluid, (4) provision of a diuretic and (5) the administration of an alkali.

The efficacy of some of these measures in experimental crush syndrome may be judged from chart 3. The intravenous administration of diuretics in doses of 0.1 mg. per gram of body weight of mannitol or sucrose (approximately 5 to 10 Gm. in a human being) or sodium sulfate (the equivalent of 1 Gm. in a patient), definitely decreased the degree of renal injury. Intravenous injection of 4 per cent gelatin solution (the equivalent in a patient of about 1 to 2 liters of plasma) further decreased renal injury and also decreased mortality. Treatment with gelatin seemed slightly more effective when the solution was made up in sixth-molar sodium lactate solution rather than isotonic solution of sodium chloride. Tolerance of crushed animals to rapid injection of gelatin was increased by the addition of calcium lactate (the equivalent of 1 Gm. in a human being).

The best result in the prevention of crush syndrome in rats was obtained by application of a pressure bandage to the injured limb within an hour after release from the crush, together with intravenous injection of gelatin in sodium lactate with added calcium lactate.

Prompt transfusion of refrigerated blood was at least as effective as infusion of gelatin (chart 1).

The problem of urinary alkalinization has been widely discussed, and most recommendations for the treatment of crush syndrome and allied states include the liberal prescription of alkali compounds, intravenously and orally, in amounts totaling 30 to 40 Gm. daily. The recommended intravenous dose of sodium citrate is 5 Gm. In crushed, myoglobin-injected rats the intravenous injection of a comparable dose of sodium citrate (0.1 mg. per gram of body weight) often caused severe tetany which was either immediately fatal or which added to the shock and renal injury, to cause delayed and excessive mortality (chart 3). Sodium bicarbonate in equal dosage, although not as immediately toxic, was equally ineffective in preventing renal injury. Thus the prevention of crush syndrome by alkalinization alone is dangerous and unjustified. The dangers of alkali treatment in these states, particularly in blackwater fever, has been recently emphasized by Maegraith and Havard[27] who justly point out that it may be impossible for an injured kidney to form alkaline urine, so that pushing alkali therapy to the point of urinary alkalinization may lead to depression of renal function[28] and dangerous alkalosis.

Our recommendations for the preventive treatment of crush syndrome and allied states are (1) pressure bandaging of the injured area and (2) prompt treatment of shock by transfusion of blood or plasma, (3) together with the infusion of 1 or more liters of sixth-molar sodium lactate and the injection of a diuretic, such as sucrose or mannitol, in repeated doses of 5 to 10 Gm. or of sodium sulfate in a dose of 1 Gm. intravenously.

Unfortunately, once the damage has been done to the kidney, the evolution of the renal injury is largely

26. Our observations indicate that provision of renal vasodilators is harmful. The aim is to prevent vasoconstriction rather than to cause vasodilation.

27. Maegraith, B. G., and Havard, R. E.: Dangers of Intensive Alkali Treatment in Blackwater Fever, Lancet **2**: 338, 1944.

28. Effect of Large Doses of Alkali on Kidney Function, Army Malaria Research Unit, Oxford, Lancet **2**: 701, 1945.

unaffected by treatment, which is supportive. Renal decapsulation has been advocated for the relief of increased interstitial pressure.[29] It is not clear that this mechanism is always significant. The value of decapsulation is not easily assessed in a disease which tends in a large number of cases to recede spontaneously at the end of one week. Histologically, the kidneys in experimental and clinical crush syndrome show extremely active regeneration of tubular cells in injured areas. Therefore, were it possible to maintain life during the period of renal injury until sufficient regeneration had occurred, it is possible that recovery would then be assured. Peritoneal dialysis[30] may answer this requirement.

SUMMARY

Post-traumatic anuria (crush syndrome) is a special instance of a group of related states of renal injury. These fall in two groups, namely, those in which the principal lesion is obstruction of the renal tubules by pigmented (myoglobin, hemoglobin) casts and those in which the injury lies principally in necrosis of the epithelium of the distal convoluted tubules. The underlying mechanism in all of these, and especially in those associated with trauma to muscle tissue, is vasoconstrictive renal ischemia. This ischemia results in oliguria, which, associated with aciduria, predisposes to precipitation of pigment casts. Tubular injury due to ischemia and a hypothetic toxic agent may contribute to the renal damage.

In the presence of such renal ischemia, myoglobinuria and hemoglobinuria, which cause little injury under normal conditions, result in severe renal injury. Attention is directed to the danger of transfusing blood which has been inadequately refrigerated.

Treatment of crush syndrome and the related states is preventive and consists in the prevention of renal vasoconstrictive ischemia by prompt treatment of shock (transfusion of blood or plasma), the lessening of absorption from areas of injury by pressure bandaging and the establishment of a rapid rate of urinary flow by the administration of a diuretic. Urinary alkalinization is advisable, but vigorous treatment with alkalies is dangerous.

ADDENDUM

Since this manuscript was submitted, it has been shown by direct means that stimulation of renal vasomotor nerves results in a change in the pattern of renal blood flow by which the cortex, which normally receives most of the renal blood flow, becomes ischemic, the stream being diverted into the medulla.[31] It has been suggested that this vasomotor mechanism may underlie the renal cortical ischemic injury of crush syndrome and allied states and that the prevention of renal injury due to this mechanism may be accomplished by procedures which block renal vasomotor pathways (splanchnic block, high spinal or caudal anesthesia).

Our failure to demonstrate an important vasomotor participation in experimental crush syndrome may result from our observations having been made in anesthetized animals. In these, by indirect means, we had shown[10] that some redistribution of cortical blood flow occurs in the renal ischemia due to bleeding or tourniquet shock. This renal ischemia was only slightly affected by renal denervation. It thus seems likely that the humoral vasoconstrictors of severe shock act on the renal circulation in a manner similar to that of intense vasomotor stimulation, i. e. by causing constriction of the cortical interlobular arteries, with cortical ischemia and diversion of blood into the medulla. Clinically, the use of procedures which block renal nerves should therefore be explored in the early treatment of crush syndrome and related states. The nature of the renal lesions does not suggest that such treatment will be of avail once renal ischemic damage and pigmentary occlusive and toxic injury are well established.

An authoritative study of crush syndrome and related renal lesions has been published by Lucké.[32]

29. Vest, S. A.: Renal Crush Syndrome, paper presented before the Section on Urology, American Medical Association, San Francisco, July 5, 1946. Peters.[25]

30. Seligman, A. M.; Frank, H., and Fine, J.: Treatment of Experimental Uremia by Means of Peritoneal Irrigation, J. Clin. Investigation **25**: 211, 1946. Abbott, W. E., and Shea, P.: Treatment of Temporary Renal Insufficiency (Uremia) by Peritoneal Lavage, Am. J. M. Sc. **211**: 312, 1946.

31. Franklin, K. J.; Barclay, A. E.; Daniel, P.; Trueta, J., and Pritchard, M. M. L.: Renal Pathology in the Light of Recent Neurovascular Studies, Lancet **2**: 239, 1946.

EXTRARENAL AZOTEMIA AND TUBULAR DISEASE

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Extrarenal azotemia may be defined as a pronounced retention of nitrogenous metabolites not caused by disease of the kidneys. The term azotemia seems preferable to uremia, since the clinical signs of uremia are not always present. This form of azotemia is not a circumscribed entity but a condition which may result from a number of unrelated causes. The essential features are oliguria or anuria, azotemia, and no anatomic lesion in the kidneys sufficient to explain renal insufficiency.

In hospitals in which routine determinations of blood urea are made during the last few days of life a surprisingly large number of patients will be found to have an elevation of blood urea to uremic levels. This report is based on a study of 84 cases of this type in which the blood urea nitrogen concentration of the patient attained a level of 50 mg. per hundred cubic centimeters or higher before death. In most instances the renal tubules show no structural changes, but often they show hydropic degeneration and, infrequently, other degenerative changes which may be partly or entirely responsible for renal failure. For this reason it is difficult to make a sharp differentiation between true extrarenal azotemia and azotemia due to tubular injury.

The cases have been arranged in several groups in accordance with etiologic factors which seem to be of major importance, but often more than one influence is present and there remains a large miscellaneous group in which the etiology has not been clearly established.

I. AZOTEMIA FROM DEHYDRATION

Azotemia from dehydration[1] (table 1) results from (*a*) decreased fluid intake, (*b*) intestinal obstruction and (*c*) excessive diarrhea.

A. *Decreased Fluid Intake.*—When an animal is deprived of water the diuresis decreases gradually and

32. Lucké, B.: Lower Nephron Nephrosis: The Renal Lesions of the Crush Syndrome, of Burns, Transfusions and other Conditions Affecting the Lower Segment of the Nephron, Mil. Surgeon **99**: 371 (Nov.) 1946.

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Read in a symposium on "Renal Insufficiency in the Shock Syndrome" before the Section on Pathology and Physiology at the Ninety-Fifth Annual Session of the American Medical Association, San Francisco, July 3, 1946.

1. Borst, J. G. G.: Acta med. Scandinav. **97**: 68, 1938; Ztschr. f. klin. Med. **117**: 55, 1931.

the urine becomes more concentrated. Finally there is complete anuria, and the blood urea increases to uremic levels. All the fluid is withdrawn from the tissue spaces, and the animal has a shrunken dehydrated appearance. The tissue spaces are emptied of fluid, but the blood will not give up any fluid to form urine when to do so would seriously reduce the blood volume.

In table 1 are tabulated 6 cases in which the azotemia was due to a decreased intake of fluid. In cases 1, 2, 3 and 5 the patients could not take fluid because of obstruction of the stomach, and in cases 4 and 6 fluid was refused because of the mental state. Only a little fluid was administered to any of these patients by the parenteral route.

In no instance of this group was there any enlargement of the kidneys, and microscopically the renal tubules were entirely normal. These 6 cases appear to be instances of true extrarenal uremia due to a decreased intake of fluid.

B. *Intestinal Obstruction.*—Dehydration from intestinal obstruction [2] (table 1) occurs most frequently from obstruction of the small intestine, less often from pyloric obstruction and paralytic ileus, and it is due in most instances to excessive loss of fluid by vomiting. So much fluid is lost in the vomitus that the patient becomes dehydrated and oliguria ensues. Chlorides are also lost excessively, and the blood chloride falls well below the normal level. For this reason the term hypochloremic uremia has been used. It was formerly believed that the loss of chloride caused retention of urea to maintain the osmotic pressure of the blood; but Kerpel-Fronius in a series of papers showed clearly that the azotemia is due to dehydration and not to low blood chloride. Experimentally he produced hypochloremia without azotemia, and he showed that in azotemia with hypochloremia the former may be corrected by the administration of water without salt. Chlorides correct the alkalosis but not the azotemia. Mach and associates found that in cirrhosis of the liver repeated drainage of the ascitic fluid reduced the blood chlorides as low as 300 mg. per hundred cubic centimeters without producing any elevation of blood urea.

In table 1 are shown 6 cases in which intestinal obstruction appears to be the major cause of the azotemia. In cases 7 to 11 there was excessive vomiting; but there was none in case 12. Possibly the accumulation of fluid in the intestinal tract may be sufficient

TABLE 1.—*Azotemia from Dehydration*

Case	Age	Sex	Disease	Blood Pressure, Mm. Hg	Blood Urea Nitrogen, Mg./100 Cc.	Specific Gravity of Urine	Weight of Kidneys, Gm.	Renal Tubules	Comment
			Subgroup A. Dehydration Due to Decreased Fluid Intake						
1	77	♂	Carcinoma of stomach	.....	65	1.029	225	Normal	Severe oliguria
2	48	♀	Lymphosarcoma of stomach	.....	65	.....	300	Normal	
3	80	♂	Carcinoma of stomach	.....	110	1.014	170	Normal	
4	75	♀	Encephalomalacia	150/ 90	87	.....	180	Normal	
5	76	♀	Carcinoma of stomach	140/100	97	.....	235	Normal	Severe ascites
6	85	♀	Encephalomalacia; pneumonia	.....	83	.....	170	Normal	
			Subgroup B. Dehydration from Intestinal Obstruction, Vomiting						
7	64	♂	Intestinal obstruction; lymphosarcoma	111/ 60	53	.....	210	Normal	
8	55	♀	Strangulated hernia	120/ 80	50 205	1.021 1.010	450	Normal	Sugical operation
9	86	♀	Ileus from impacted gallstone	Very low	181	.....	315	Normal	Low blood pressure
10	82	♂	Paralytic ileus	120/ 70	69	1.032	195	Normal	Severe oliguria
11	61	♂	Carcinoma of stomach	.....	129	1.018	372	Normal	
12	63	♂	Appendicitis; ileus	.....	198	.....	390	Normal	
			Subgroup C. Dehydration from Diarrhea						
13	2 mo.	♀	Mongolism	.....	81	.....	30	Normal	CO_2 combining power 12 vol. %
14	59	♀	Carcinoma of stomach	.....	61	.....	320	Hydropic, 3	
15	50	♂	Chronic ulcer; colitis	.....	78	.....	265	Normal	

TABLE 2.—*Azotemia from Low Blood Pressure*

Case	Age	Sex	Disease	Blood Pressure, Mm. Hg	Blood Urea Nitrogen, Mg./100 Cc.	Specific Gravity of Urine	Weight of Kidneys, Gm.	Renal Tubules
16	36	♂	Lobar pneumonia	90/50	102	.....	355	Normal
17	30	♂	Hemorrhage, rupture of liver	60/ ?	96	.....	330	Hydropic, 1
18	63	♀	Coronary sclerosis	80/55	80	1.022	310	Hydropic, 1
19	43	♀	Acute hemorrhagic pancreatitis	70/40	50	.....	280	Normal
20	62	♂	Perforated duodenal ulcer	98/52	55	.....	330	Normal
21	64	♂	Coronary sclerosis	75/60	80	1.023	415	Normal
22	79	♂	Carcinoma of colon	84/50	110	.....	336	Normal
23	84	♂	Carcinoma of cecum	Very low	57	1.020	205	Normal
24	60	♂	Aortic stenosis	80/62	96	1.024	340	Hydropic, 1
25	81	♂	Pneumonia	70/40	60	.....	226	Normal
26	30	♂	Lobar pneumonia	90/61	71	.....	230	Normal
27	70	♀	Coronary thrombosis	90/70	61	.....	290	Normal
28	66	♂	Carcinoma of colon	60/ ?	51	.....	300	Normal
29	47	♂	Cerebral thrombosis	78/58	92	.....	300	Normal
30	86	♀	Intestinal obstruction	68/40	56	.....	180	Normal
31	55	♂	Multiple fractures	84/ ?	64	.....	360	Hydropic, 1
32	29	♂	Multiple fractures	70/ ?	68	.....	280	Hydropic, 1
33	40	♂	Fractures of pelvis and femur	70/30	88	1.022	510	Normal
34	40	♂	Fracture; peritonitis	70/30	72	.....	347	Hydropic, 1
35	84	♀	Fracture of femur; pneumonia	.....	60	.....	194	Normal

2. (*a*) Kerpel-Fronius, E.: Ztschr. f. d. ges. exper. Med. **97**: 733, 1936. (*b*) Mach, R. S.; Mach, E., and Scicioumoff, F.: Schweiz. med. Wchnschr. **64**: 54, 1934.

to cause dehydration of the tissues. In case 9 the low blood pressure of the patient was probably contributory to the oliguria and azotemia. In case 8 the kidneys were swollen, but in no instance in this group were there any structural alterations in the renal tubules.

C. *Excessive Diarrhea.*—In Asiatic cholera the dehydration, oliguria and azotemia are due to loss of fluid through the intestine. In table 1 3 cases are shown in which the azotemia was attributed to excessive loss of fluid from diarrhea. In cases 13 and 15 the renal tubules are normal, but in case 14 there is a severe hydropic degeneration of the tubules (fig. 1). In this instance it appears that the tubular injury is as important as the loss of fluid in causing the azotemia.

Unfortunately there are only a few observations on the specific gravity of the urine in the dehydration group, but they are perhaps sufficient to show that the urine is not always as concentrated as one might expect. It has been noted in several instances of azotemia due to dehydration that the administration of large quantities of fluid parenterally may increase the diuresis without increasing the specific gravity of the urine or lowering the level of the blood urea. These observations suggest that the renal tubules are injured in dehydration azotemia, but they showed structural changes in only 1 of 15 cases.

TABLE 3.—*Postoperative Azotemia*

Case	Age	Sex	Disease	Blood Pressure, Mm. Hg	Blood Urea Nitrogen, Mg./100 Cc.	Specific Gravity of Urine	Weight of Kidneys, Gm.	Renal Tubules	Comment
36	64	♂	Carcinoma of pancreas	.....	94	.....	465	Hydropic, 2	Severe oliguria
37	28	♀	Carcinoma of rectum	108/65 80/ ?	130	.....	360	Hydropic, 2	Severe oliguria
38	61	♂	Hypertrophy of prostate	104/70 40/ ?	110	.....	240	Necrosis	Severe oliguria; pneumonia
39	49	♀	Umbilical hernia	.....	82	.....	685	Normal	Pneumonia
40	82	♂	Hypertrophy of prostate	88/50 60/38	163	.....	250	Normal	Anuria
41	45	♂	Carcinoma of rectum	.....	202	1.012	555	Hydropic, 2	Severe oliguria
42	50	♀	Myomas of uterus	.....	53	.....	336	Normal	Paralytic ileus; vomiting
43	58	♀	Carcinoma of stomach	85/68	61	.....	290	Hydropic, 3	Some necrosis of the liver
44	52	♂	Gangrene of small intestine	High	132	.....	500	Hydropic, 1	Severe oliguria
45	25	♂	Retrograde pyelograms	.....	...	.....	440	Normal	Reflex anuria; edema of lungs

TABLE 4.—*Miscellaneous Cases of Extrarenal Azotemia*

Case	Age	Sex	Disease	Blood Pressure, Mm. Hg	Blood Urea Nitrogen, Mg./100 Cc.	Specific Gravity of Urine	Weight of Kidneys, Gm.	Renal Tubules	Comment
46	74	♂	Hypertension, myocardial failure	165/115	67	1.018	220	Normal	Diarrhea
47	57	♂	Myocardial failure	.....	75	.....	873	Normal	
48	74	♂	Coronary sclerosis	108/ 94	53	1.006 1,025	260	Normal	
49	57	♂	Hypertension, coronary sclerosis	132/ 64	125	1.022	355	Normal	
50	79	♀	Hypertension, myocardial failure	220/110	52	.....	335	Normal	
51	60	♂	Coronary sclerosis	.....	173	1.021	350	Normal	Diabetes
52	50	♂	Old valve defect	118/ 70	85	.....	298	Normal	Severe oliguria
53	40	♂	Bacterial endocarditis	126/ 70	155	1.016	435	Normal	
54	27	♂	Rheumatic endocarditis	.....	135	1.020	275	Normal	
55	36	♀	Empyema	100/ 80	92	.....	440	Normal	
56	60	♂	Pneumonia	180/ 90	58	1.020	335	Normal	
57	82	♂	Pneumonia	.....	102	.....	285	Hydropic, 1	
58	88	♂	Pneumonia	104/ 64	104	.....	225	Normal	
59	54	♀	Pneumonia	.....	102	.....	175	Normal	
60	75	♀	Empyema	100/ 80	78	.....	260	Hydropic, 2	
61	87	♀	Pneumonia	.....	66	.....	230	Hydropic, 1	
62	65	♂	Pneumonia	100/ 60	67	.....	252	Normal	
63	68	♂	Pneumonia	108/ 60	104	1.018	250	Hydropic, 2	Severe oliguria, sulfadiazine
64	50	♂	Typhoid fever	144/ 90	70	.....	385	Hydropic, 1	Sulfadiazine
65	51	♀	Streptococcic septicemia	112/ 80	75	.....	350	Normal	
66	74	♀	Acute cholecystitis	.....	55	.....	198	Normal	
67	46	♀	Pelvic suppuration	130/ 75	56	.....	240	Normal	
68	67	♀	Encephalomalacia	.....	60	.....	202	Normal	
69	68	♀	Encephalomalacia	180/110	82	.....	225	Normal	
70	73	♀	Encephalomalacia	130/ 90	93	1.030	255	Normal	
71	45	♂	Delirium tremens	125/ 76	80	.....	294	Normal	
72	55	♀	Lymphosarcoma, jaundice	145/ 85	54	.....	250	Hydropic, 1	
73	67	♂	Carcinoma of lung	120/ 90	54	1.013	305	Normal	
74	58	♀	Carcinoma of gallbladder	130/ 90	66	.....	210	Hydropic, 1	
75	77	♀	Carcinoma of stomach	.....	73	1.021	170	Normal	
76	42	♂	Carcinoma of liver	140/ 60	100	.....	500	Bile casts	
77	53	♀	Cirrhosis of liver; jaundice	96/ 56	66	.....	345	Normal	
78	42	♂	Cirrhosis of liver	116/ 70	50	.....	370	Normal	
79	64	♂	Addison's disease	90/ 65	57	.....	290	Normal	
80	70	♂	Burns (25 days)	.....	52	1.023	310	Hydropic, 1	
81	61	♀	Diabetic coma	.....	123	1.024	265	Normal	
82	33	♀	Diabetic coma	90/ 85	59	1.019	380	Normal	
83	62	♂	Diabetic coma	110/ 60	68	.....	265	Normal	
84	67	♂	Necrosis of liver	152/ 78	175	.....	440	Normal	Lobar pneumonia

II. AZOTEMIA FROM LOW BLOOD PRESSURE

In the second group (table 2) there are listed 20 cases in which the azotemia was probably due in large measure to low blood pressure.[3] In each instance the

3. Evans, T. S.: Azotemia with Normal Kidneys Found Post Mortem, Arch. Int. Med. **48**: 1231 (Dec.) 1931.

blood pressure was extremely low during the last day or two of life. When the systolic pressure falls to 80 mm. of mercury or lower glomerular filtration is reduced and the glomerular filtrate is almost entirely reabsorbed in the tubules. There is also the factor of anoxia associated with the decreased flow of blood to the renal tissue. This is the condition which prevails in shock and all these patients were in a state of shock terminally. In case 17 the shock was due to a massive intraperitoneal hemorrhage. It will be noted that in 5 cases, 31 to 35, the shock was due to fractures.

Fig. 1 (case 14, table 1).—Severe hydropic degeneration of the renal tubules.

In 14 cases the renal tubules appeared normal histologically, but in 6 instances there was a mild hydropic degeneration, which suggests that tubular injury may have been a contributory cause of the oliguria and consequent azotemia.

III. POSTOPERATIVE AZOTEMIA

The third group, postoperative azotemia [4] (table 3), is not a well-defined group from the point of view of causation, since more than one influence may be concerned. One factor which is often operative is postoperative shock with a fall of blood pressure. There may also develop some complication such as pneumonia or ileus. It is important to note that 5 of the 10 cases listed in table 3 showed hydropic degeneration of the renal tubules indicative of tubular injury (fig. 2). In case 43 the hydropic degeneration was severe, and there were small areas of necrosis in the liver.

Reflex oliguria: Occasionally after surgical operations, especially on the ureter, kidney, bladder, lower colon and gallbladder, severe oliguria and azotemia develop. The renal function prior to operative intervention is normal, there is no postoperative fall of blood pressure, and the kidneys are not traumatized. The blood urea may rise to uremic levels. The diuresis is usually reestablished after a few days of anuria, and operative intervention is not indicated. It has been suggested that the suppression of urine is due to overactivity of the sympathetic nervous system, such as occurs in paralytic ileus.

In case 45 anuria developed in the patient following bilateral retrograde pyelograms. In order to start a diuresis large quantities of fluid were given intravenously. The patient died on the third day from edema of the lungs.

IV. MISCELLANEOUS CASES OF EXTRARENAL AZOTEMIA

The fourth group constitutes miscellaneous cases of extrarenal azotemia, which occurs in the terminal stages of a large variety of diseases. These have been grouped to some extent in table 4. In the cases of heart disease [5] (cases 46 to 54) low blood pressure was apparently not a factor, and the fluid intake was satisfactory. The urine was not highly concentrated, but the renal tubules were normal on histologic examination. No cause of the azotemia is apparent.

In the cases of pneumonia and empyema (cases 55 to 63) hydropic degeneration of the tubules was found in 4 instances, but in 1 of these (case 63) sulfadiazine may have been responsible. There is, however, a suggestion that suppuration may produce tubular injury.

In this miscellaneous group the cause of the azotemia is not clear. In patients with a satisfactory fluid-intake and a normal blood pressure azotemia may develop during the last few days of life. This terminal azotemia occurs frequently. It appears that 25 per cent of persons dying of such diseases as lobar pneumonia and cancer show blood urea at uremic levels on the last day of life. It has been suggested that increased destruction of tissue proteins is partly responsible for this terminal uremia. It is important to remember that

Fig. 2 (case 37, table 3).—Hydropic degeneration of the renal tubules indicative of tubular injury.

4. Bannick, E. G., and Keith, N. M.: M. Clin. North America 11: 1571, 1928.

5. Kartagener, M.: Klin. Wchnschr. 1: 1028, 1933.

a terminal rise of blood urea does not indicate a primary renal disease.

Azotemia is a frequent complication of diabetic coma[6] (cases 81 to 83). About twenty-four to forty-eight hours after the onset of coma oliguria or anuria develops. Under treatment the patient may recover from the coma and the hyperglycemia and acidosis may be corrected, yet the anuria may persist. At autopsy the kidneys are frequently swollen, but the microscopic changes are hardly sufficient to explain the anuria. It has been suggested that the tubules are injured from the acidosis or the low blood pressure, and that the disturbance is of a functional nature.

OTHER FORMS OF EXTRARENAL AZOTEMIA

There are other forms of extrarenal azotemia which are not represented in the 84 cases here reported.

Azotemia from Hemorrhage into the Gastrointestinal Tract.—Massive hemorrhage into the gastrointestinal tract,[7] usually from bleeding ulcers, causes an increase of blood urea sometimes to uremic levels. There is oliguria and sometimes anuria. The urine may show a good concentration of urea, but the specific gravity is not high (around 1.022) and may be as low as 1.012. The several factors to which this form of azotemia has been attributed are: absorption of blood from the gastrointestinal tract, low blood pressure and decreased blood volume.

Borst attributed azotemia to the rapid absorption of urea formed from the blood in the intestinal tract, and this interpretation has been adopted by others. However, Johnson gave 1,200 to 1,500 cc. of blood to 4 different patients by stomach tube during a period of six to eight hours. The blood urea rose only moderately and transiently, except in 1 patient whose urea clearance was only 50 per cent of normal, in which instance it reached 54 mg. per hundred cubic centimeters. The absorption of blood would help to explain the azotemia but not the oliguria.

It appears more probable that the azotemia is due to the combined effect of low blood pressure, which is often present, and the dehydrating effect of the hemorrhage. After a hemorrhage, fluid is withdrawn from the tissue spaces to restore the blood volume and none is available for the formation of urine.

The Crush Syndrome.[8]—Bywaters, Beall and others have reported over 20 cases of oliguria and azotemia following crushing injuries of an extremity sustained during air raids. On admission the patient is usually in good condition except for the swelling of the crushed extremity. The extremity usually does not become gangrenous, but some of the muscle is necrotic. The hemoglobin is increased and the patient is thirsty, indicating a loss of fluid into the swollen extremity, as in a severe burn. Shock may develop, but this does not always occur. Diuresis becomes progressively less, hypertension and azotemia develop and the patient may die of uremia, or recovery may follow the onset of diuresis. Some of the patients pass a dark-colored urine which gives a positive benzidine reaction and shows the bands of myohemoglobin on spectroscopic examination. At autopsy the kidneys are swollen and edematous. The investigators attribute the azotemia chiefly to tubular injury, especially injury of the distal convoluted tubules. The structural changes in the tubules seem hardly sufficient to explain the anuria, yet the clinical evidence indicates that their function is seriously impaired.

Paxson and associates described 3 cases of urinary suppression: 1 from a retroplacental hematoma, 1 from rupture of the uterus and 1 from infarction of a cystic ovary. They regard these as similar to the crush syndrome.

Azotemia from Subarachnoid Hemorrhage.—In occasional instances of subarachnoid hemorrhage[9] blood urea is increased to uremic levels. This is a transitory phenomenon and no satisfactory explanation is available (Romcke and Skouge).

COMMENT

Extrarenal azotemia may be brought about by several different mechanisms, and the pure extrarenal types cannot be separated sharply from urinary suppression due to tubular injury. Anuria and oliguria are symptoms of severe tubular injury, and there can be no doubt that tubular injury is responsible for urinary suppression in such diseases as mercuric chloride poisoning and some cases of anuria following sulfonamide therapy and blood transfusions, in which striking degenerative changes in the tubules are visible. In 20 of the 84 cases on which this study is based there was mild to severe hydropic degeneration of the proximal convoluted tubules, indicating that tubular injury may have been in part or entirely responsible for the uremia. In 2 of these 20 cases sulfadiazine was probably the cause of the injury to the tubules. In the other 64 cases there were no demonstrable changes in the tubules. When the tubules appear normal histologically one may believe that their function is also normal, or it may be maintained that functional failure may occur without alterations of structure that we are able to recognize.

It seems clearly established that low blood pressure and dehydration of any type may produce azotemia, and since neither of these mechanisms involves failure of the tubules one does not expect to find tubular degeneration. Likewise in reflex anuria the tubules are not primarily concerned.

The terminal azotemia which occurs so frequently in a large variety of diseases cannot be explained entirely as due to dehydration and low blood pressure. An increased tissue catabolism during the last days of life may be partly responsible for the increased level of urea in the blood. Toxic substances derived from necrotic and inflammatory tissues may injure the renal tubules sufficiently to cause oliguria without producing structural alterations that can be recognized.

The specific gravity of the urine has not been studied sufficiently, and our observations are inadequate. The few determinations that were made indicate that the urine is seldom concentrated even in the presence of

6. Baker, T. W.: Clinical Survey of One Hundred and Eight Consecutive Cases of Diabetic Coma, Arch. Int. Med. **58**: 373 (Sept.) 1936. Christensen, O., and Holst, J. E.: Ztschr. f. klin. Med. **111**: 88, 1929. Dinkin, L.: Klin. Wchnschr. **6**: 1330, 1927. Fullerton, H. W., and others: Lancet **1**: 558, 1932.

7. Alsted, G.: Am. J. M. Sc. **192**: 199, 1936. Bookless, A. S.: Guy's Hosp. Rep. **88**: 22, 1938. Campbell, D. C., and Stickney, J. M.: Proc. Staff Meet., Mayo Clin. **17**: 145, 1942. Christiansen, T.: Acta med. Scandinav. (supp.) **78**: 894, 1936. Gregory, R.; Ewing, P. L., and Levine, H.: Azotemia Associated with Gastrointestinal Hemorrhage, Arch. Int. Med. **75**: 381 (June) 1945. Johnson, J. B.: J. Clin. Investigation **20**: 161, 1941. Jung, G.: Ztschr. f. klin. Med. **118**: 546, 1931. Meyler, L.: Acta med. Scandinav. **87**: 313, 1935.

8. Beall, D.; Bywaters, E. G. L.; Belsey, R. H. R., and Miles, J. A. R.: Brit. M. J. **1**: 432, 1941. Bywaters, E. G. L.: Ischemic Muscle Necrosis, J. A. M. A. **124**: 1103 (April 15) 1944. Bywaters, E. G. L., and Beall, D.: Brit. M. J. **1**: 427, 1941. Bywaters, E. G. L., and Dible, J. H.: J. Path. & Bact. **54**: 111, 1942. Helwig, F. C., and Orr, T. G.: Traumatic Necrosis of Liver with Extensive Retention of Creatine and High Grade Nephrosis, Arch. Surg. **24**: 136 (Jan.) 1932. Mayon-White, R., and Solandt, D. M.: Brit. M. J. **1**: 434, 1941. Paxson, N. F.; Golub, L. J., and Hunter, R. M.: Crush Syndrome in Obstetrics and Gynecology, J. A. M. A. **131**: 500 (June 8) 1946.

9. Morawitz, P., and Schloss, J.: Klin. Wchnschr. **11**: 1628, 1932. Romcke, O., and Skouge, E.: Acta med. Scandinav. **77**: 211, 1931.

severe oliguria, which indicates that the process is not one of simple dehydration. When the diuresis is low and the glomerular filtrate moves slowly through the tubules it is possible that more of the crystalloids are reabsorbed or that more urea diffuses back into the blood. Much could be learned from chemical study of the urine.

The creatinine increases in the blood corresponding with the increase of urea. Extrarenal uremia cannot be distinguished from uremia of renal origin by the creatinine level.

The few observations available indicate that the carbon dioxide combining power of the plasma decreases as the blood urea level rises, as in renal uremia.

SUMMARY

Eighty-four cases of terminal azotemia are reported in which the blood urea nitrogen ranged from 50 to 205 mg. per hundred cubic centimeters. In 20 cases there was mild to severe hydropic degeneration of the proximal convoluted tubules indicating that tubular injury may have been partly responsible for the azotemia.

A terminal rise of blood urea nitrogen to uremic levels occurs in a high percentage of persons and in a number of different diseases.

A high blood urea nitrogen level on the last day of life does not mean that the patient has a primary renal disease.

In these 84 cases the azotemia is attributed to dehydration in 15, to low blood pressure in 20 and to postoperative complications in 10 cases. Three cases were associated with diabetic acidosis and 1 with necrosis of the liver. In the remaining 35 cases the cause of the azotemia could not be established.

ABSTRACT OF DISCUSSION

ON PAPERS OF DR. MOON, DRS. MARTINEAU AND HARTMAN, DRS. CORCORAN AND PAGE AND DRS. BELL AND KNUTSON

DR. E. T. BELL, Minneapolis: Our knowledge of extrarenal uremia is incomplete. We know that dehydration and low blood pressure account for many cases, but for the majority we have no adequate explanation. Dr. Corcoran has shown that some cases are due to vasoconstriction. Apart from these three forms, we still have a large group of cases, especially those that come on during the last few days of life, about which we have practically no ideas. The blood pressure is normal, and the patient is well hydrated, and yet uremia develops. The kidneys appear normal. Work on this problem has really just begun. We must try to find out why the uremia develops. Is it entirely the fault of some mechanism outside the kidneys, or do the kidneys really play a role? Better methods are needed for study of the histology of the tubules. Unless the tissue is fresh and the fixation good even normal tubules appear to be damaged, and many pathologists have been misled by artefacts. We also need to know what functional changes take place in the kidneys in extrarenal azotemia. Why cannot the kidneys concentrate the urine in the presence of oliguria? These are some of the points on which we would like to have more information.

DR. A. C. CORCORAN, Cleveland: This symposium has established the occurrence in a wide variety of diseases of a common clinical and functional pattern of renal damage characterized by tubular necrosis and degeneration in association with variable numbers of pigmented casts. In some cases the casts are few and apparently noncontributory, and in others they are many and a major factor in the renal damage. The lesions which occur in the absence of any large number of pigmented casts we would view as due primarily to renal vasoconstriction of humoral origin and consequent anoxia. The deposition of pigment may be added to this. Our experience indicates that even minor degrees of vasoconstrictive renal ischemia greatly facilitate the intratubular deposition of pigment. In treatment, urinary alkalinization by the intravenous injection of sodium citrate is often recommended. However, such an injection is often lethal in rats with crushed limbs, apparently by addition of tetany to preexisting shock. If alkalinization is to be attempted, and there is much to suggest its value, it should be accomplished cautiously and by injection of sodium lactate rather than sodium citrate.

DR. VIRGIL H. MOON, Philadelphia: I wish only to reemphasize one or two items. Vasoconstriction is certainly a factor in many instances; in all forms of secondary shock, vasoconstriction is one of the compensatory mechanisms which the body has called into play to sustain the circulation at an adequate level, and that vasoconstriction in itself may produce anoxic changes in the kidney. These may be so severe as to cause degenerative changes in the tubules. Some of the cases which it was my privilege to study were those in which aviators at high altitudes, supplied with oxygen masks, succumbed to anoxia by some accident happening to the oxygen apparatus. Perhaps moisture froze within the mask; perhaps the lines were cut by flak or otherwise. Those who died of acute anoxia—death probably occurred within fifteen minutes from the time when lack of oxygen became pronounced—showed parenchymatous degeneration of the kidneys. This was of the same type but not of the same degree as that seen in the cases of longer duration. In regard to terminology: it is not the physician's function to attach names to things, but it would be advantageous if this item which has been rediscovered recently by physiologists, pathologists and internists should receive a designation. It would be historically appropriate if this were called acute parenchymatous nephritis, although it is not truly an inflammatory condition as would be implied by "itis." Other appropriate terms would be acute nephrosis or acute tubular disease. The cases described could accurately be termed acute tubular disease.

DERMATITIS DUE TO QUINACRINE HYDROCHLORIDE ("ATABRINE")

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Pasadena, Calif.

The purpose of this presentation is to recapitulate the cutaneous manifestations which occur following the administration of quinacrine hydrochloride ("atabrine *di*-hydrochloride" N. N. R.). In the past eight months numerous articles have been presented or published, chiefly dealing with the lichenoid type of cutaneous reaction. This type has been variously described as atypical hypertrophic lichen planus, New Guinea disease or tropical lichen planus. Since practically all the published observations on this type of reaction are in accord, it will be considered only briefly; although exceedingly interesting to dermatologists, it comprises only a small percentage (about 12 per cent) of the dermatoses produced by this drug.

Soon after the beginning of the New Guinea campaign it became apparent that cutaneous disorders were a prolific source of trouble in the Southwest Pacific area. Many conflicting ideas were expressed. The average soldier soon came to call every cutaneous eruption "jungle rot." The medical officer tended to ascribe the conditions to fungi, especially when the hands, feet and groin were involved. Contact dermatitis became a favorite diagnosis as more and more troops came into intimate contact with the profuse vegetation of the jungle. Sensitivity to sunlight or a pellagroid reaction was considered in those cases in which the eruption first appeared on the exposed areas of the body. Impetigo, pyoderma and erysipeloid eruption became favorite diagnoses in one group of these cases. The otologist

Read before the Section on Dermatology and Syphilology at the Ninety-Fifth Annual Session of the American Medical Association, San Francisco, July 5, 1946.

encountered many cases of otitis externa, which he generally considered as tropical mycosis. If a little dandruff was present a diagnosis of seborrheic dermatitis was entertained. Cases of generalized exfoliative dermatitis, severe and not infrequently fatal, began to appear in persons who had not received arsenical drugs. All of these factors, combined with the injudicious topical use of the sulfonamide compounds, ammoniated mercury and ointment of benzoic and salicylic acid, carbolfuchsin paint and other similar remedies, together with the effects of a tropical climate, produced a picture that was indeed confusing. As more cases were seen and various factors considered, it became apparent that there was only one in common, namely quinacrine hydrochloride. In the course of time it became possible to classify these cutaneous reactions in three groups: the eczematoid, the hypertrophic lichenoid and the generalized exfoliative.

First is the eczematoid group, which comprises about 80 per cent of the cases and which was overlooked by all except those who had seen service in the tropical areas of the South Pacific.[1] This reaction is characterized by a chronic, dry or exudative, symmetric, localized or generalized eczematous eruption. It is extremely recalcitrant to treatment, and its course generally is marked by periods of remission and exacerbation, though it may be steadily progressive. The sites of predilection are the ears, eyelids, periorbital region, the dorsa of the hands and feet and those areas most often affected in seborrheic dermatitis. On involution peculiarly pigmented flat or hypertrophic lichenoid lesions may develop at the sites of healing lesions. In some cases the eruption appears primarily as a pyogenic infection in the bearded region, the scalp and eyebrows or in the pubic area. On involution these lesions often produce a patchy alopecia. An alopecia totalis may occur.

The second group constitute the lichenoid reactions. This is the type which was most familiar to dermatologists in this country and Australia and about which a great deal has been written. Actually this group of reactions comprises only about 12 per cent of the total incidence of this cutaneous complex. While the eruption in these cases may closely resemble, both morphologically and histologically, that of a hypertrophic lichen planus, it has a more widespread and different distribution, often involving face and trunk or the palms and soles. Involvement of the nails is common, often producing changes similar to those of psoriasis or mycotic infections. In other cases there is a slate-blue pigmentation of the nail bed. Extensive lesions resembling leukoplakia or those seen in lichen planus are common on the mucous membranes of the mouth. Pigmented lesions without apparent changes in the texture of the mucous membranes are frequently seen in the same location. The eruption elsewhere varies from flat pigmented macules, in which the texture of the skin is normal, to thick, confluent, nodular, hyperkeratotic or verrucous lesions, the color of which ranges from pink to a deep violaceous hue.

The third group is the exfoliative reaction, which presents a severe exfoliative dermatitis resembling that due to the arsenobenzols. Secondary infection is common, and some fatalities occurred before penicillin became available. This type is occasionally primary but frequently secondary to the lichenoid or eczematoid forms.

1. Novy, F. G., Jr.. Personal communication to the author.

INCIDENCE

Since all information concerning the untoward effects of quinacrine hydrochloride was suppressed during the war, no accurate statistics as to the actual incidence of dermatitis due to this drug are available. However, it was unquestionably high, as is shown by the following facts: In 1943, when there were comparatively few troops stationed in the Southwest Pacific, this theater of action alone accounted for 66 per cent of the total number of patients evacuated from all theaters of war on account of diseases of the skin. Practically all these cases originated in New Guinea, where "atabrine" discipline was rigidly enforced. In 1944 20 per cent of hospital admissions in New Guinea and a slightly lower percentage of evacuations of noneffective persons to the United States were on account of dermatologic conditions. At the same time it was commonly found that as high as 50 per cent of the men reporting for sick call in unit dispensaries did so on account of diseases of the skin. It is my opinion and that of other observers that from 80 to 90 per cent of these cases, though classified under various diagnoses, were in reality cases of dermatitis due to quinacrine hydrochloride.

CONTROLS

Due to the fact that exacerbation did not occur immediately on readministration of quinacrine hydrochloride, there are those who have doubted the role of this drug in the disorder. However, there are a large number of controls present, if one wishes to look for them. These are as follows:

1. American prisoners of war in the Philippines. It is a singular fact that in all the several thousand Americans who were prisoners of the Japanese after Bataan and Corregidor there has not been 1 instance of the cutaneous disorder described in this paper. These prisoners were on starvation diets which produced the wet and dry forms of beriberi. They suffered from pellagra, dysentery and malaria. Further, they lived, perspired and worked in a tropical climate. It is exceedingly strange that these "x" factors of some of the authors were unable to produce the entity previously described in the absence of the ingestion of quinacrine hydrochloride.

2. Military personnel. In troops and Navy personnel serving under identical conditions in the malaria-free areas of the tropical South Pacific where no quinacrine hydrochloride was administered, instances of this entity were not encountered.

3. Japanese prisoners of war. Until the proper authorities publish their reports on the number, physical condition and incidence of skin diseases of the Japanese taken prisoner by the American forces, no reliable data can be given. It is my impression that the Japanese soldier under identical conditions of tropical warfare but without benefit of quinacrine hydrochloride did not present this cutaneous symptom complex.

4. Mediterranean theater of operations. It has been stated that this symptom complex did not occur in the Mediterranean area even though certain units were in malaria-infected districts and the troops were taking quinacrine hydrochloride. However, this is not the case, and several examples of the lichenoid types of eruption occurring in British soldiers have been presented before the Section on Dermatology of the Royal Society of Medicine, although the causative factor of

quinacrine hydrochloride was not recognized.[2] It has also been reported from every other theater of war where this drug was used as a suppressive for malaria.

CONTACT DERMATITIS

One case of contact dermatitis of the eyelids in a nurse handling quinacrine hydrochloride has been reported.[3] The cases of contact dermatitis, rhinitis, bronchitis and asthma in pharmaceutic workers, as reported by Watrous,[4] are of interest in this regard. He stated that in a large laboratory in over 70 per cent of the men who handled the dry drug these conditions developed within a period of eight months. He further stated that "the potency of quinacrine as a local irritant is such that even with elaborate dust control equipment . . . a wide variety of symptoms were observed in the workmen."

OTHER UNTOWARD EFFECTS OF QUINACRINE HYDROCHLORIDE

Any drug producing such severe cutaneous reactions as does quinacrine hydrochloride must of necessity also cause damage elsewhere in the body. Thus it is known that hepatic damage occurs, which may end in death.[5] It is not without significance that most cases of severe hypoplastic anemia in troops in the Southwest Pacific area have been associated with so-called atypical lichen planus.[6] Acute quinacrine intoxication has been reported.[7] Disturbance of vision with advanced impairment of eyesight has been attributed to this drug.[8] The entire question of psychosis occurring in troops under suppressive therapy with this agent needs further study and reexamination in the light of recent work.[9] It is of interest, though uninformative, to note that there has been only one communication dealing with the untoward effects of the drug in pharmaceutical and manufacturing plants.[4]

MECHANISM OF PRODUCTION OF REACTIONS

Those immediate reactions to quinacrine hydrochloride, such as generalized pruritus, urticaria or exfoliative dermatitis, which occur in a small percentage of persons shortly after beginning treatment with the drug, can be considered allergic in character. Also, the scarlatiniform and morbilliform eruptions occurring in the first few days of treatment can be explained on a biotrophic basis and considered as examples of Milian's ninth day erythema. However, in the vast majority of cases in which the appearance of the eruption is delayed for many weeks or months after beginning the routine administration of quinacrine hydrochloride it can hardly be attributed to either an allergic or biotrophic reaction. The known cumulative effect of the substance with its long retention in the body; the hepatic damage, hypoplastic anemias, inanition, gastrointestinal symptoms, mental disturbances and transient paralysis which frequently accompany the cutaneous manifestations, all strongly suggest a toxic reaction. This would also account for the negative results obtained in intradermal and patch tests and for the frequent delay in the exacerbation of the eruption after the readministration of the drug.

Incidentally, the newer synthetic antimalarial (chloroquine SN 7618[10]), which is apparently a better drug than either quinine or quinacrine, is known to produce lichenoid lesions.

In view of the great difficulty in explaining the causation of quinacrine dermatitis by any of the present theories of drug-induced eruptions, it would seem that the entire problem of their etiology might well be subjected to critical reexamination.

TREATMENT

Preventive.—Owing to its cumulative effect, in a large percentage of normal persons toxic manifestations will eventually develop from even small doses of quinacrine hydrochloride if it is given over a long period. For this reason, the successful use of this drug as either a suppressive or therapeutic agent in malaria is entirely dependent on the early recognition of these toxic symptoms and the immediate discontinuance of the drug. In patients receiving this agent, the appearance of any lichenoid or eczematoid eruption, a pruritus or an urticaria, however slight, should be considered as a warning signal and as potentially dangerous. In these cases the drug should be discontinued immediately, quinine being substituted if necessary. All other actively toxic drugs such as arsenic, mercury, gold or sulfonamide compounds are contraindicated. Extension of the eruption or exfoliative dermatitis may follow the use of the sulfonamide drugs either locally or internally. Two cases of the relatively benign lesions of the lichenoid type were seen in which a fatal aplastic anemia followed the intramuscular injections of a bismuth or mercury compound.

Eczematoid Type.—The treatment in the eczematoid types of dermatitis is that of any other irritable dermatosis. Only the blandest local medicaments should be used. If moist dressings are required, weak solutions of potassium permanganate, aluminum acetate or isotonic solution of sodium chloride are indicated. Soothing lotions, such as Mook's or calamine, are useful in the moist eczematous forms, and simple ointments in the dry scaly types. The secondary infection, which may be a serious problem in these cases, should be controlled by the use of penicillin, given either locally or by intramuscular injection. It is of great value, especially in the severe generalized exfoliative or eczematoid types. It should be continued in small maintenance doses of from 80,000 to 90,000 units until the infection disappears. Untoward reactions to penicillin used in this manner were extremely infrequent in a large series of cases, and none were of a serious type. The results of penicillin therapy are particularly spectacular in the severe exfoliative forms, greatly reducing the nursing care required by rendering unnecessary the prolonged tub baths and continuous moist dressings formerly used.

2. Hellier, F. F., in discussion on Dowling, G. B.: Erythematous Lichen Planus, Proc. Roy. Soc. Med. **37:** 410 (June) 1944; Brit. J. Dermat. **56:** 241 (Nov.-Dec.) 1944; Two Cases of Lichen Planus, Proc. Roy. Soc. Med. **37:** 413 (June) 1944; Brit. J. Dermat. **56:** 245 (Nov.-Dec.) 1944.

3. Whitehill, R.: Skin Sensitivity Due to Atabrine, Bull. U. S. Army M. Dept. **4:** 724 (Dec.) 1945.

4. Watrous, R. M.: Quinacrine Dihydrochloride: Occupational Disorders and Their Control, Indust. Med. **13:** 887 (Oct.) 1944.

5. Drake and Moon: Read at the meeting of the Section of Dermatology and Syphilology of the California Medical Association, May 1946.

6. Most, H., and Hayman, J. M., Jr.: Recovery from Severe Hypoplastic Anemia Associated with Atypical Lichen Planus, Bull. U. S. Army M. Dept. **5:** 339 (March) 1946.

7. Burnham, R. C.: Acute Atabrine Intoxication, U. S. Nav. M. Bull. **46:** 434 (March) 1946.

8. Chamberlain, W. P., Jr., and Boles, D. J.: Corneal Edema: Quinacrine (Atabrine), Bull. U. S. Army M. Dept. **5:** 6 (Jan.) 1946. Abbey, E. A., and Lawrence, E. A.: Effect of Atabrine Suppressive Treatment on Eyesight of Pilots, ibid. **5:** 130 (Feb.) 1946.

9. Gaskill, H. S., and Fitz-Hugh, T., Jr.: Toxic Psychosis Following Atabrine, Bull. U. S. Army M. Dept., March 1945, no. 86, p. 63. Lerro, S. J.: Report of Two Cases of Toxicity to Atabrine, Mil. Surgeon **89:** 668 (Oct.) 1941. Mergener, J. C.: Psychosis Following the Administration of Quinacrine Hydrochloride for Malaria, War Med. **8:** 250 (Oct.) 1945. Crawford, R. P.: Atabrine Psychosis, Air Surgeon's Bull. **2:** 21 (Jan.) 1945.

10. Loeb, R. F., and others: Activity of a New Antimalarial Agent, Chloroquine (SN 7618), J. A. M. A. **130:** 1069 (April 20) 1946.

Lichenoid Type.—The lichenoid type of reaction requires little treatment other than the cessation of the drug and the usually supportive measures, such as a high caloric and high vitamin diet. If pruritus is present the use of mild antipruritic ointments is indicated. In recalcitrant cases superficial roentgen therapy is of value.

CONCLUSIONS

1. Quinacrine hydrochloride ("atabrine *di*-hydrochloride" N. N. R.) is the etiologic agent in a large group of cutaneous conditions occurring in troops serving in the tropics.

2. The eczematoid type of reaction to this drug is the commonest and has been overlooked to a large extent.

3. The lichenoid type of reaction presents a bizarre picture of peculiarly pigmented, slate-blue to purplish hypertrophic lichenoid lesions.

4. The exfoliative type of reaction may occur primarily or as a progression of the eczematoid or lichenoid types.

5. In the mechanism of the production of this cutaneous syndrome, none of the generally accepted theories of drug-evoked eruptions appear adequate to explain the observed facts.

ABSTRACT OF DISCUSSION

DR. JAMES R. DRAKE, San Francisco: I should like to compliment Dr. Nisbet not only on his paper but on his early recognition of an important group of cases in the early phases of military activities in the southwest Pacific. It has been somewhat difficult to prove the role of quinacrine hydrochloride in this regard from a strictly scientific point of view. Many clinical workers in both the Army and Navy have attempted to produce exacerbations with readministration of the drug, with varying results. In my group, the eczematoid type flared fairly promptly and with a fair degree of regularity, while the lichenoid did not. Ingestion of the drug over a longer period of time was apparently necessary. Similarly, the antimalarial was taken for a longer period of time prior to the onset of the lichenoid type. The average time of taking the drug prior to the onset in my group of lichenoid cases, some 75 consecutive cases, was about five and a half months. Other workers have reported anywhere from two to six months as their average. It should be kept in mind also that cutaneous reactions to this drug antedated World War II and occurred in civilians. Holton and Winchester in 1938 reported a severe case of exfoliative dermatitis, presumably due to quinacrine hydrochloride. I believe this was the first case reported in the United States. That a contact dermatitis may occur was demonstrated by Dr. George Andrews before the New York Dermatological Society in 1943 with a case of a chemist exposed to quinacrine dust. Since that time contact sensitivity has, as Dr. Nisbet stated, been repeatedly demonstrated, both in the production of the drug and by patch testing a number of workers in both branches of the armed services. In a group of cases observed by Dr. Moon of Letterman General Hospital, during an eighteen month period, 9 patients with lichenoid dermatitis died. In 7 of these the cause of death was aplastic anemia. During the same eighteen months, only a total of 9 cases of aplastic anemia were seen at Letterman General Hospital. On the other hand, in this group of cases it was impossible to demonstrate any definite connection with hepatic damage during the same period of time.

DR. FREDERICK G. NOVY JR., Oakland, Calif.: Dr. Nisbet has given us an excellent discussion of the various forms of eruptions produced by the ingestion of quinacrine hydrochloride. The hypertrophic form is so spectacular that diagnosis clinically is not hard to make. The eczematoid type, in my opinion, is nearly impossible to diagnose clinically. While I was in the Navy, a number of cases of eczematoid dermatitis occurred in the tropics among the personnel who had been aboard ship or who were in some nonmalarious area, where they were not getting this drug. Clinically it was impossible to differentiate these cases. I think they can only be diagnosed when one has a positive history and in addition exacerbate the eruption by giving the drug. During the middle part of the war, this disease was called the New Guinea disease. The reason it was seen in New Guinea in such high incidence was because there was a large concentration of troops that had been there for a long time and the "atabrine" discipline was very strict. The result was the appearance of many cases of this eruption in that area. Since then other cases have been reported from various surrounding islands. There have been cases, as Dr. Nisbet mentioned, from the Mediterranean theater, India and elsewhere. As the war progressed, we began to see patients who had gone directly to the Philippines in whom the eruption developed while there. Toward the end of the war, the patients were seen who had gone directly from the United States to Okinawa, which is 26 degrees north and out of the tropics. So I feel that the climate and environment of New Guinea itself did not play a role in the production of this spectacular and unusual disease. My experience has been the same as that of Drs. Drake and Nisbet in regard to exacerbations of the lesions in patients. These eruptions do not exacerbate with just the administration of one or two tablets of quinacrine hydrochloride. The eczematoid type usually takes six or seven days or a little longer to develop, and a hypertrophic type usually takes considerably longer than does the eczematoid.

COMMANDER E. E. BARKSDALE, Bethesda, Md.: When investigation of this problem was first ordered at Bethesda it was called New Guinea lichen planus. It was possible to reproduce the condition in Washington, D. C. It is admitted that the climate in Washington is probably worse than it is in New Guinea. But this is a definite quinacrine-caused dermatitis. When 200 cases for investigation were requested after the Surgeon General had ordered the investigation, in only 38 of the 200 cases was it possible to prove true dermatitis caused by this drug, and that was from the entire Navy and Marine Corps in the Pacific. My impression is that it is a rare disease. Seventeen patients were selected to be put back on a regimen of quinacrine hydrochloride; in 16 of the 17 it was possible to reproduce the disease. In the other patient, administration of the drug was discontinued because of nausea and vomiting. I think it is a fixed type of drug eruption due to quinacrine hydrochloride.

DR. MARVIN KNOLL, Los Angeles: I have seen this form of dermatitis from Milne Bay in New Guinea up through the Philippines and on the island of Honshu at Kure, Osaka and Kobe. All the patients gave a history of previous medication with quinacrine hydrochloride. Dr. T. Tanimura, professor of dermatology at the Imperial University in Osaka, told me he had not encountered a like condition among the Japanese troops. In Papua, New Guinea, from January 1944 until July 1945, a similar dermatitis was not seen in hospitalized or well natives. Among American service men and women the variety of cases ranged from small split-pea-sized individual lesions to the severe universal exfoliative dermatitis complicated by blood dyscrasias. Atabrine contains the acridine nucleus, a photosensitizing radical, and I believe many of these cases had their early beginning as a result of the sensitization of the skin to sunlight.

DR. ALFRED HOLLANDER, Springfield, Mass.: Several months ago I saw a case which differs somewhat from the cases reported by the physicians who were in New Guinea. This patient while in the Pacific had taken quinacrine hydrochloride for thirty months up to November 1945 without any ill effects. He returned to this country in December 1945; he was first in the south, and he came back to Massachusetts in March 1946, when he had his first attack of malaria. He was again given quinacrine hydrochloride for about ten days. About three days after the discontinuance of the drug he had a yellow discoloration of the entire skin surface. I saw him about a week later, at which time he still showed the yellow discoloration and, furthermore, two slightly papular lesions the size of a nickel in the region of the right elbow. I told him to report back if he should see any further outbreak. He did

not return until four weeks later, when he exhibited a lichen-planus-like eruption on various parts of his body, which I could not differentiate from any outbreak of typical lichen planus. In regard to Dr. Knoll's remarks, in Massachusetts there were cases of dermatitis due to this drug at Fort Devens in 1944. I think these cases with pigmentary changes should be compared with the cases Riehl of Vienna described at the end of World War I and which are called Riehl's melanosis. At that time it was a nutritional factor which brought out the disease. I believe that in dermatitis due to quinacrine hydrochloride a nutritional factor together with the climate and the sensitivity to the drug produces the pigmentary changes.

RHEUMATIC BRAIN DISEASE

Late Sequel of Rheumatic Fever

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In recent years attention has been directed to the occurrence of rheumatic obliterating arteritis in the brain of persons with rheumatic heart disease.[1] This late cerebral sequel of rheumatic fever involves mainly the small meningeal and cortical vessels, producing gross or microscopic softenings in the cortex. Less frequently, larger vessels are affected. The involvement of the brain may occur from several months to many years after the acute stage of rheumatic fever, at a time when the person is otherwise enjoying good physical health. To this hitherto unrecognized cerebral process the term rheumatic brain disease was given. Similar cases have since been described in this country [2] and abroad.[3]

Although the frequency with which widespread and clinically manifest arteritis takes place in the brain of persons with rheumatic cardiovalvular disease is not yet known, its occurrence is by no means rare. It has been estimated from necropsy experience that approximately 5 per cent of the patients of the average mental hospital are afflicted with a combined rheumatic brain disease and rheumatic heart disease.

In 85 per cent of these patients the sedimentation rate was found to be accelerated. Correlative histologic studies disclosed rheumatic activity on the valves of the heart and in the cerebral and other vessels, indicating the presence of subclinical rheumatic fever in the absence of other symptoms which are generally regarded as manifesting activity.[1]

From the Research Department, Central State Hospital, and the Department of Psychiatry and Neurology, Indiana University School of Medicine.

Read before the Section on Nervous and Mental Diseases at the Ninety-Fifth Annual Session of the American Medical Association, San Francisco, July 3, 1946.

1. Bruetsch, W. L.: Psychosen bei chronischer Endokarditis, Psychiat.-neurol Wchnschr. **38**: 551 (Oct. 31); 563 (Nov. 7) 1936; The Histopathology of the Psychoses with Subacute Bacterial and Chronic Verrucose Rheumatic Endocarditis, Am. J. Psychiat. **95**: 335 (Sept.) 1938; Chronische rheumatische Gehirnerkrankung als Ursache von Geisteskrankheiten: Eine klinisch-anatomische Studie, Ztschr. f. d. ges. Neurol. u. Psychiat. **166**: 4, 1939. Bruetsch, W. L., and Bahr, M. A.: Chronic Rheumatic Brain Disease as a Factor in the Causation of Mental Illness, J. Indiana M. A. **32**: 445 (Sept.) 1939. Bruetsch, W. L.: Chronic Rheumatic Brain Disease as a Possible Factor in the Causation of Some Cases of Dementia Praecox, Am. J. Psychiat. **97**: 276 (Sept.) 1940; Rheumatic Epilepsy: Sequel of Rheumatic Fever, ibid. **98**: 727 (March) 1942; Rheumatic Endarteritis of Cerebral Vessels: Sequel of Rheumatic Fever, Tr. Am. Neurol. A. **68**: 17, 1942; Late Cerebral Sequelae of Rheumatic Fever, Arch. Int. Med. **73**: 472 (June) 1944; Sedimentation Rate and White Blood Count in Mental Patients with Rheumatic Brain Disease, paper read at the annual meeting of the American Psychiatric Association, Chicago, May 28, 1946; Am. J. Psychiat., to be published.

2. Kernohan, J. W.; Woltman, H. W., and Barnes, A. R.: Involvement of the Nervous System Associated with Endocarditis, Arch. Neurol. & Psychiat. **42**: 789 (Nov.) 1939. Dublin, W. B.: Pathologic Lesions of the Brain Associated with Chronic Rheumatic Endocarditis and Accompanied by Psychosis, Dis. Nerv. System **2**: 390 (Dec.) 1941.

3. Castex, M. R.: Rheumatismo cerebral crónico, Prensa méd. argent. **30**: 1401 (Aug. 4) 1943.

REPORT OF CASE

History and Course.—A woman patient aged 49 had four slight strokes, which were followed by a change in personality. After the last cerebral vascular accident, which affected her speech, she became destructive and uncontrollable.

The past history mentioned a leakage of the heart, which was discovered at the age of 20. There was no definite history of rheumatic fever in childhood. When the patient was 35 years of age, she had been in bed for several months with sciatic rheumatism.

Physical examination revealed a diastolic murmur at the apex. The pulse was irregular both in time and force. Its volume was small. The rate was 80 per minute. The blood pressure was 115 systolic and 90 diastolic. The roentgenogram and the electrocardiogram were indicative of mitral stenosis. An impediment of speech and hyperactive patellar reflexes were the only neurologic abnormalities. Mentally, the patient exhibited a considerable degree of deterioration. Pneumoencephalography showed dilatation of the right lateral ventricle. There was an increased amount of cortical air over both hemispheres. The widening of the subarachnoid spaces was particularly well defined in the region of the right sylvian point.

The Wassermann reaction of the blood and spinal fluid was negative. The cell count, total protein determination and colloidal gold curve of the spinal fluid were normal. Urinalysis

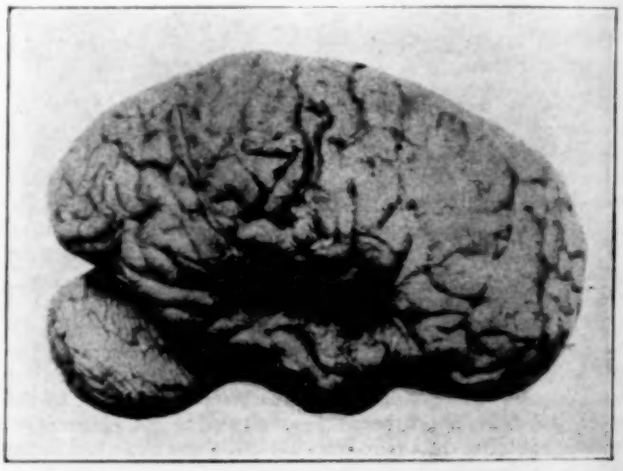

Fig. 1.—Rheumatic brain disease. Cortical infarction in right temporal lobe due to rheumatic obliterating arteritis.

revealed a trace of albumin and granular casts. There was an increased rate of sedimentation on several occasions, oscillating between 18 and 22 per hour (Cutler vein method). The temperature was normal. The blood count was as follows: hemoglobin 72 per cent; red cells 3,800,000; leukocytes 8,950. The differential count indicated a shift to the left.

The following diagnoses were made: psychosis with rheumatic brain disease; mitral stenosis, and subclinical rheumatic fever. The patient remained in the institution for four years. At no time was there any sign of congestive heart failure. She died suddenly from a pulmonary infarct.

Postmortem Observations.—Necropsy revealed rheumatic mitral stenosis. The mitral valve was thickened in its entire circumference. The chordae tendineae were shortened. Aschoff bodies were not found in the cardiac muscle, but isolated Aschoff cells were scattered throughout the myocardial interstitium. It has been said that Aschoff nodules are present only in about 11 per cent of rheumatic patients who die in the later decades of life.[4]

The lungs revealed recent rheumatic vascular disease accompanied by red infarcts. Numerous Masson bodies, which are considered the equivalent of Aschoff bodies in the heart, were observed in the pneumonic tissue.[5] In both kidneys were old

4. Console, A. D.: Relation of Cardiac Lesions to the Clinical Course of Rheumatic Fever, Arch. Int. Med. **69**: 551 (April) 1942.

5. Neubuerger, K. T.; Geever, E. F., and Rutledge, E. K.: Rheumatic Pneumonia, Arch. Path. **37**: 1 (Jan.) 1944.

infarctions, which could be traced to rheumatic obliterating endarteritis of renal vessels.[6]

In the right temporal lobe (fig. 1) of the brain was a large area of cortical softening, which extended into the anterior limb of the internal capsule. Smaller infarctions were present in the central and parietal cortex of both sides. Extensive

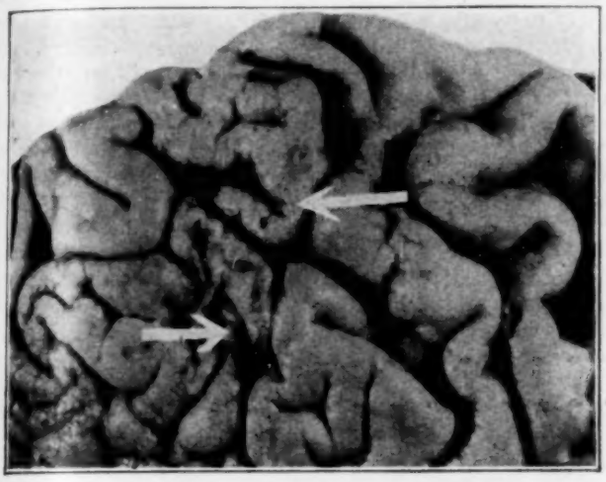

Fig. 2.—Softening and granular atrophy of convolutions in rheumatic brain disease.

histologic search detected occluded meningeal vessels, which were responsible for the cortical softenings. The rheumatic vascular obliterations extended over short segments only and for this reason could easily have been missed unless serial sections were made. In addition to the gross infarcts there were present in the gray matter of all lobes microscopic areas of incomplete softening (acellular areas). In the vessels of the brain not a single yellow plaque was noted, excluding arteriosclerosis as the cause of the cerebral changes.

In the spinal cord were minor rheumatic alterations, consisting of incomplete vascular occlusions without softenings. There

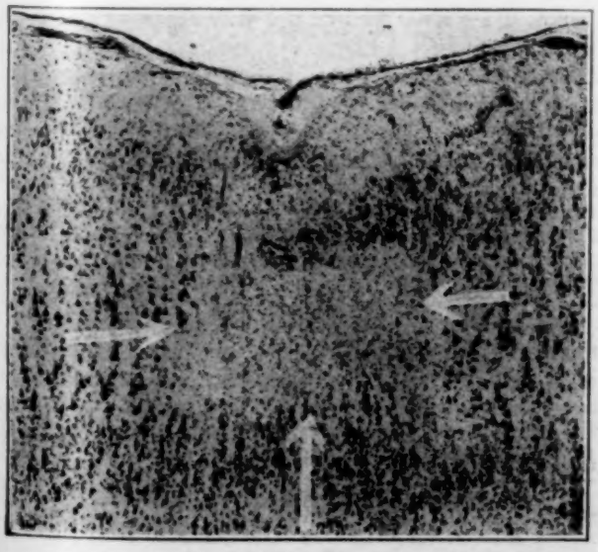

Fig. 3.—Microscopic area of incomplete softening (acellular area) in cerebral cortex; toluidine blue stain.

were also a few vessels which were surrounded by lymphocytes. These lesions, however, were not sufficient to produce such clinical symptoms of myelitis as were observed by Mørch Christensen in the wake of rheumatic fever.[7]

6. Hutton, R. L., and Brown, C. R.: The Renal Lesion in Rheumatic Fever, Ann. Int. Med. **20**: 85 (Jan.) 1944.
7. Mørch Christensen, A.: Myelitis Developing as Sequel of Rheumatic Fever, Hospitalstid. **81**: 327 (April 3) 1938.

COMMENT

This patient with rheumatic mitral stenosis, while in other respects in good health, experienced at the age of 49 a rheumatic involvement of the brain in the form of a diffuse obliterating process of the cerebral vessels. Similar vascular changes were observed in the kidneys. Furthermore, rheumatic fever was still or was again active in the lungs of this patient, as evidenced by Masson bodies and an increased rate of sedimentation, although the initial infection must have taken place several decades earlier. The disease history in this case exemplifies the validity of the newer concept which looks on rheumatic fever as a generalized process, involving various organs by way of the vascular system and being a continuous infection which lasts throughout life in some persons.

SYMPTOMATOLOGY OF RHEUMATIC BRAIN DISEASE

Rheumatic disease of isolated larger vessels of the central nervous system may produce various obscure neurologic conditions, which have puzzled the older neurologists. Leyden [8] in 1877 reported the sudden development of bulbar palsy in a woman aged 52, who

Fig. 4.—Mitral valve with recurrent rheumatic endocarditis. There is a large verruca on the closing border. To the right is a continuous but barely visible layer of verrucous tissue.

at the age of 22 had had an acute form of rheumatic fever. There was mitral stenosis and a small area of fresh softening in the medulla oblongata. Vascular occlusions in patients with rheumatic endocarditis have in the past been interpreted as always being caused by the detachment of emboli from the cardiac valves. In the vast majority of patients, however, such occlusions, in the absence of auricular fibrillation, are the result of local proliferation of the intima.

In general, rheumatic endarteritis has a predilection for the smaller vessels and in the brain involves frequently only the small meningeal and cortical branches, resulting in microscopic areas of softening in the gray matter. The ensuing mental disturbance is determined by the personality and by the age at which the cerebral involvement occurs. If the rheumatic infection causes cerebral damage in childhood, aberrations ranging from mild behavior disorders to slight mental deficiency states may result. In the young adult psychopathologic pictures suggesting dementia precox have been observed. Although the active rheumatic state is generally regarded as a disease of childhood, the late vascular lesions of rheumatic fever are not infrequently seen in middle life and even in the aged, being responsi-

8. Leyden, E.: Zwei Fälle von acuter Bulbärparalyse, Arch. f. Psychiat. **7**: 44, 1877.

ble for psychoses characteristic of that period. This is in accordance with the more recently emphasized observation that in many adult cases of rheumatic heart disease an active infection persists, usually in a manner so mild as to be scarcely detectable.[9]

RHEUMATIC BRAIN DISEASE AS A FACTOR IN THE CAUSATION OF SOME CASES OF DEMENTIA PRECOX

The studies on rheumatic brain disease have unearthed the fact that at necropsy 9 per cent of patients with dementia precox showed signs of rheumatic infection involving the brain as well as the cardiac valves.[1]

Modern psychiatry has viewed dementia precox and mental disease in general as a mere maladjustment of the person to social, environmental and emotional situations. The mass experiments which tested the so-called dynamic theories of psychiatry were the experiences of the two world wars. In environmental or emotional stresses were the cause of disabling mental illness, there would have been a decided increase of dementia precox and of other psychoses during the two wars. This was not the case.[10]

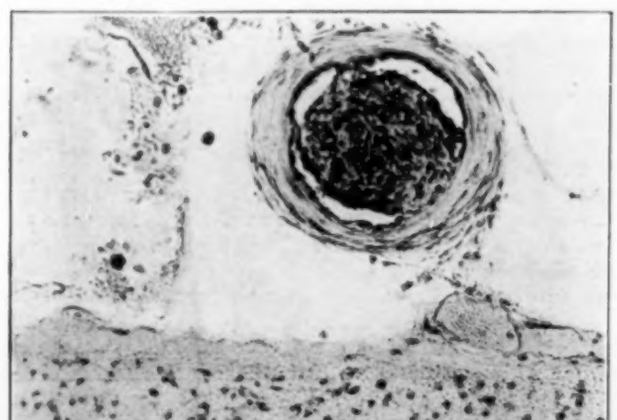

Fig. 5.—Rheumatic obliterating endarteritis of small meningeal artery. Proliferated intimal cells fill the lumen of the vessel. In the underlying cortex the ganglion cells have disappeared or show signs of degeneration; toluidine blue stain.

At various times the view has been expressed by Wagner-Jauregg,[11] Bleuler,[12] Singer[13] and others[14] that dementia precox does not represent an etiologic entity but is a clinical syndrome capable of subdivisions.

It seems as if the description of rheumatic brain disease, which occurs in 9 per cent of schizophrenic patients, is the first successful attempt to break down the dementia precox group into subgroups with definite etiologies.[1]

A few cases in which this cerebral sequel of rheumatic fever produced the symptomatology of dementia precox will be described.

A woman at the age of 35 began to hear voices which threatened her life. Later she had periods during which she became combative and violent. Except for a history of antecedent rheumatic fever, little was known about her life. She died after eleven years of institutional confinement in a state of advanced dementia. In the brain were gross (fig. 2) and microscopic (fig. 3) infarctions, which were due to rheumatic endarteritis of meningeal and cortical vessels. There were many old vascular occlusions, pointing out that one was not dealing with a terminal process. The clue to the exact nature of the cerebral involvement was furnished by the mitral valve, which disclosed chronic rheumatic endocarditis (fig. 4).

The history of another patient is revealing.

The woman patient, aged 37, was admitted to the Central State Hospital in 1890, coming from another institution where she had been an inmate since the age of 25. The diagnosis on admission was, according to the classification then in use, chronic mania. In 1915 the diagnosis was changed to dementia precox, and in 1925 the more modern term of schizophrenia was attached to the patient. When she died, examination of the brain and organs brought to light the true reason why this patient had spent the greater part of her life in a mental institution. In the brain was widespread rheumatic endarteritis (fig. 5) of many years' standing, associated with numerous microscopic areas of incomplete softening. In the kidneys were the residue of old rheumatic infarcts. The mitral valve divulged rheumatic alterations.

It may be interjected here that the rheumatic cerebral damage in these patients was not the anatomic substratum of the psychosis but was only an incidental and unrelated observation. The same inference could be made in regard to psychoses with vascular neurosyphilis. However, few clinicians doubt that the syphilitic obliterations of the blood vessels with their resulting cortical necroses constitute the basis of the mental disorder and are not merely terminal lesions.

Furthermore, the rare opportunity had presented itself to examine the brain of a schizophrenic patient with rheumatic endocarditis, who five months after the onset of the psychosis committed suicide. Here the same changes characteristic of rheumatic brain disease were present.

DIFFERENTIAL DIAGNOSIS BETWEEN RHEUMATIC BRAIN DISEASE AND CEREBRAL LESIONS IN THROMBOANGIITIS OBLITERANS (BUERGER'S DISEASE)

An increasing number of cases with cerebral involvement in thromboangiitis obliterans (Buerger's disease) is being reported.[15] In some of these patients,[16] however, endocarditic changes on the cardiac valves have been present, implying that the vascular alterations represented rheumatic arteritis and not thromboangiitis

9. Juster, I. R.: The Significance of Rheumatic Activity in Chronic Rheumatic Heart Disease, Am. Heart J. **15:** 1 (Jan.) 1938.

10. Stokes, A. B.: War Strains and Mental Health, J. Nerv. & Ment. Dis. **101:** 215 (March) 1945.

11. Wagner-Jauregg, J.: Was' leistet die hormonale Behandlung bei einer im Klimakterium aufgetretenen Schizophrenie? Med. Welt **9:** 1605 (Nov. 2) 1935.

12. Bleuler, E. P.: The Physiogenic and Psychogenic in Schizophrenia, Am. J. Psychiat. **87:** 203 (Sept.) 1930.

13. Singer, H. D.: Psychosis and the Central Autonomic Nervous System, J. A. M. A. **110:** 2048 (June 18) 1938.

14. Sherman, I. C., and Broder, S. B.: Dr. H. Douglas Singer's Concept of the Psychoses, Arch. Neurol. & Psychiat. **49:** 732 (May) 1943. Bumke, O.: Die Auflösung der Dementia Praecox, Klin. Wchnschr. **3:** 437 (March 11) 1924. Bouman, K. H.: De schizophrenie als organische hersenziekte, Nederl. tijdschr. v. geneesk. **75:** 2564 (May 16) 1931. Dunlap, C. B.: Dementia Praecox: Some Preliminary Observations on Brains from Carefully Selected Cases, and a Consideration of Certain Sources of Error, Am. J. Psychiat. **80:** 403 (Jan.) 1924.

15. Foerster, O., and Guttmann, L.: Cerebrale Komplikationen bei Thrombangiitis obliterans, Arch. f. Psychiat. **100:** 506, 1933. Hausner, E., and Allen, E. V.: Cerebrovascular Complications in Thrombo-Angiitis Obliterans, Ann. Int. Med. **12:** 845 (Dec.) 1938. Lindenberg, R., and Spatz, H.: Ueber die Thromboendarteriitis obliterans der Hirngefässe (cerebrale Form der v. Winiwarter-Buergerschen Krankheit), Virchows Arch. f. path. Anat. **305:** 531, 1939. Davis, L., and Perret, G.: Cerebral Thromboangiitis Obliterans: Report of Three Verified Cases, Tr. Am. Neurol. A. **68:** 12, 1942. Scheinker, I. M.: Cerebral Thromboangiitis Obliterans: Histogenesis of Early Lesions, Arch. Neurol. & Psychiat. **52:** 27 (July) 1944.

16. Spatz, H.: Ueber die Beteiligung des Gehirns bei der v. Winiwarter-Buergerschen Krankheit (Thrombo-endangiitis obliterans), Deutsche Ztschr. f. Nervenh. **136:** 86, 1935. Sträussler, E.; Friedmann, R., and Scheinker, I.: Ueber die Endangiitis obliterans (v. Winiwarter-Buergersche Krankheit) unter besonderer Berücksichtigung der Hirnveränderungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **160:** 155, 1937. Krayenbühl, H.: Zur Diagnostik und chirurgischen Therapie der zerebralen Erscheinungen bei der Endangiitis obliterans v. Winiwarter-Buerger, Schweiz. med. Wchnschr. **75:** 1025 (Nov. 29) 1945.

obliterans. It is obvious, then, that a diagnosis of Buerger's disease in the presence of rheumatic cardiovalvular disease should be made with caution. Even the histologic differentiation between thromboangiitis obliterans and rheumatic vascular disease is at times most difficult.[17] A general criterion is that Buerger's disease involves particularly the larger arteries, while rheumatic fever has a preference for the smaller vessels.

CONCLUSIONS

About 5 per cent of the patients in mental hospitals have a combined rheumatic brain disease and rheumatic heart disease. The ratio between rheumatic men and women is 1 : 3.

The sedimentation rate was accelerated in 85 per cent of these patients, suggesting the presence of subclinical rheumatic fever.

The cerebral involvement consisted of a rheumatic obliterating endarteritis with subsequent areas of softening. To this process the term rheumatic brain disease was given.

ABSTRACT OF DISCUSSION

DR. FREDERICK P. MOERSCH, Rochester, Minn.: Dr. Bruetsch has added another chapter to his many studies on the involvement of the nervous system associated with rheumatic heart disease. That cases of fatal rheumatic heart disease reveal definite pathologic findings in the brain has also been substantiated by other investigators. As a result of all these studies there is no longer any good reason to doubt the close relation between rheumatic heart disease and the many neurologic disorders that accompany it. That cerebral lesions of rheumatic origin may produce mental symptoms seems a reasonable deduction. Here the correlation between the pathologic observations and the clinical symptoms is not as clear as it is in the cases of a neurologic nature. Unfortunately it is not known how numerous or how extensive lesions in the brain must be for the production of mental symptoms. It is known that senile plaques may be found in the brain without any apparent associated mental symptoms; however, if the number of plaques is extensive there are the mental symptoms of Alzheimer's disease or senile dementia. In 1939 Dr. Kernohan and his associates (*Arch. Neurol. & Psychiat.* **42**:789-809 [Nov.] 1939), in a study of the "Involvement of the Nervous System Associated with Endocarditis," found neuropathologic changes similar to those noted by Dr. Bruetsch. In 23 of their 42 cases there was rheumatic endocarditis with fatal outcome. In 21 of these 23 there were neuropsychiatric disorders, and in 5 there was evidence of psychosis. In spite of these observations it remains a fact that from a clinical point of view mental symptoms are seldom observed in patients with rheumatic heart disease. Many patients are seen who have rheumatic heart disease; they are seen repeatedly and in various stages of their illness, but it is rare indeed to find any unusual mental symptoms which one might attribute to rheumatic brain damage. To my knowledge, no extensive psychiatric study has been carried out in patients suffering from rheumatic heart disease. Certainly such a clinical study seems indicated, as undoubtedly more careful psychiatric surveys would probably indicate a much greater incidence of psychiatric symptoms than is now appreciated.

DR. NATHAN MALAMUD, San Francisco: Dr. Bruetsch has for a number of years drawn attention to his observations of rheumatic brain disease in a variety of neurologic and psychiatric disorders. There is one aspect of this problem which I would like to discuss, namely, the value of such studies in conditions which are usually dismissed as being functional, particularly in schizophrenia. In this disorder organic cerebral changes are denied, but, as Dr. Bruetsch has emphasized, investigation in routine state hospital autopsies might disclose subgroups in which an organic process is undeniable. The vascular disease reported by Dr. Bruetsch probably forms one of these subgroups. There is another in which a similar mechanism, though of different etiology, might play a role. This concerns some patients with catatonic schizophrenia in whom, at the height of an acute attack, signs of vasomotor instability, convulsions and other organic symptoms develop, with fatal termination. In such cases the observations at necropsy are invariably negative. However, careful microscopic investigation of the brain shows signs of impaired cerebral circulation but without evidence of any vascular disease or occlusion. Two cases will serve as illustrations. A catatonic form of schizophrenia of about one and a half years' duration in a woman aged 20 ended abruptly in an episode of stupor, convulsions and hyperthermia which lasted three days, pathologically showing extensive infarction of the cerebral cortex in the presence of normal blood vessels. In the cortex of this patient there were numerous acellular areas and small infarcts at an early stage, but the meningeal and cortical vessels themselves were normal and not occluded. In the second case recurrent catatonic attacks over a number of years in a man aged 24 terminated in an episode of stupor associated with hyperthermia, bradycardia and apnea and pathologically showed widespread changes of the cortex without any vascular lesions. In this second case there was distinct paling of the cortex in some areas, particularly the troughs of the convolutions by contrast with the crests. On higher magnification there was widespread ischemic degeneration of the ganglion cells. It may be significant that the same mechanism of disturbed cerebral circulation is suggested in these so-called functional disorders as in the cases of rheumatic brain disease. I should like to ask Dr. Bruetsch whether he has any explanation for the occurrence of schizophrenic, epileptic or other types of disorder in the different cases he reported, in spite of apparently similar pathologic conditions in the brain.

DR. N. W. WINKELMAN, Philadelphia: The late Dr. John Eckel of Buffalo and I reported in 1932 a group of 5 cases from a great number of similar cases from the Philadelphia General Hospital, in which the brain of the patient was involved in acute rheumatic fever. To this condition we gave the name of nonsuppurative encephalitis rheumatica. From our study we concluded that acute rheumatic fever does not produce a specific change in the brain that can be recognized either grossly or microscopically. Second, the changes that are present in the brain of patients who have shown neurologic and psychiatric symptoms during the course of acute rheumatic fever are similar in every way to the changes that occur in any other acute infections and toxemias. We considered that edema of the brain played a part in the production of the acute manifestations. We found an endarteritis of the small vessels similar to that described in syphilis by Nissl and Alzheimer, which we felt might be the result either of a mechanical process, the edema, or of a toxic reaction through the blood stream. At that time we found the same acellular areas in the acute cases that Dr. Bruetsch observed in his chronic cases, but I must add also that we found the same areas absolutely indistinguishable from those of rheumatic fever patients with other infections and toxemias. We felt that the occurrence of endarteritis completely changed the picture in the brain. We found so-called brain purpura, hemorrhagic encephalitis, in a great many of our cases of endarteritis. I would like to ask Dr. Bruetsch first, if he has found similar pathologic changes in patients with histories of other infectious processes and second, whether he believes the endarteritis itself is responsible for the changes that he has described in the brain.

DR. J. M. NIELSEN, Los Angeles: Does Dr. Bruetsch think that the actual picture of dementia precox or schizophrenia is ever produced by these rheumatic lesions, inasmuch as dementia precox begins with a loss of interest and attention, seclusiveness and so on, followed only much later by symptoms referable to the cortex? I do not see how any type of pathologic anatomy in the cortex or subcortex can ever explain the entire syndrome

17. Jäger, E.: Zur pathologischen Anatomie der Thrombangiitis obliterans bei juveniler Extremitätengangrän, Virchows Arch. f. path. Anat. **284**: 526 and 584, 1932. Fahr, T.: Die rheumatische Granulomatose (rheumatisches Fieber, Rheumatismus infectiosus specificus, Rheumatismus verus) vom Standpunkt des Morphologen, Ergebn. d. inn. Med. u. Kinderh. **54**: 357, 1938.

of dementia precox. There are many symptoms in dementia precox that are referable to the diencephalon, the thick subcutaneous tissue, the oiliness of the skin, the general unhealthy appearance of the patient in general and a certain peculiar pathologic plantar response which is not a Babinski. Things of that sort characterize dementia precox, and it seems to me that they are not simulated exactly by any other condition of the brain.

Dr. Leopold Hofstatter, St. Louis: Dr. Bruetsch's paper is an important contribution toward a better understanding of schizophrenic reactions in relation to physical illness. In a number of brains from schizophrenic patients he could show cortical changes that he identified as late sequels of rheumatic fever. The significance of the pathologic conditions described for the etiology of schizophrenic reactions will have to be evaluated in the light of similar observations in mentally healthy persons. The fact that personality features often are a determining factor in the final outcome of physical illness may have some bearing on the matter. From Dunbar's pertinent studies it is known that rheumatic fever in the repressed, passive and submissive person usually results in a well-established, irreparable pathologic process (rheumatic heart disease) whereas the more spontaneous and aggressive patient recovers without permanent (cardiac) damage. The occurrence of organic changes of the brain as sequels of rheumatic fever in schizophrenics may therefore be explained on psychosomatic grounds.

Dr. Walter L. Bruetsch, Indianapolis: I want to answer first the question of Dr. Moersch, who mentioned that there are many patients with rheumatic heart disease who do not manifest mental symptoms. In this connection there is an analogy with syphilis. There are many instances of syphilis, but in only a few patients does there develop vascular neurosyphilis (syphilitic endarteritis) associated with mental symptoms. Dr. Malamud portrayed the cerebral changes of patients whose disease was diagnosed during life as dementia precox, emphasizing the point, which also was the theme of my paper, that in some cases of dementia precox there are demonstrable organic lesions. Dr. Winkelman brought up the question whether the vascular alterations are specific for rheumatic fever. I am inclined to the statement that the etiology of obliterating endarteritis at times cannot be diagnosed from the morphologic appearance of the lesion alone. Various conditions lead to endarteritis, among them syphilis, thromboangiitis obliterans and rheumatic fever. In the study with which this paper is concerned only cases which also involved rheumatic endocarditis were considered. This established with certainty the rheumatic etiology of the vascular process. For some time internal medicine has recognized endarteritis due to rheumatic fever. It has been shown to occur in almost every organ of the body. Finally, the cerebral vessels have been added. Dr. Winkelman in 1929 published a number of instances with proliferative endarteritis of varied causation, and in 1 of these cases the vascular occlusions were obviously due to rheumatic fever. As to the question whether the symptoms of dementia precox could be caused by the cerebral changes which were observed in these patients, no single localization or type of cerebral process is recognized which will produce the symptomatology of dementia precox. This possibly answers to some extent Dr. Nielsen's question. In general one may say, however, that a diffuse involvement of the cerebral cortex is one of the various possibilities which can initiate schizophrenic symptoms. The description of rheumatic brain disease directs attention to a hitherto unrecognized phase of rheumatic fever. This has led to concern among persons with a history of rheumatic fever and among parents whose children have this disease. There is, however, a note of optimism. Even if the brain should become involved in later years, rheumatic encephalopathy has a fairly high rate of spontaneous recovery. The recovery may occur even though gross or microscopic cerebral lesions have occurred. This may be explained by the fact that after the acute vascular process in the cerebral vessels has subsided, there remains enough normal brain tissue for the patient to continue mental processes. In some instances recovery has been speeded by intensive salicylate therapy.

Clinical Notes, Suggestions and New Instruments

GRANULOCYTOPENIA DUE PROBABLY TO "PYRIBENZAMINE"

WYNDHAM B. BLANTON, M.D.
and
M. E. B. OWENS Jr., M.D.
Richmond, Va.

Granulocytopenia is believed not to have been described hitherto among the untoward side effects of tripelennamine hydrochloride, "pyribenzamine" (N. N. R.) (N'pyridyl-N' benzyl-N-dimethylethylenediamine hydrochloride), therapy.

REPORT OF CASE

A widow aged 73, whose general health had always been excellent, was first seen in 1942 for a vulvovaginitis. She was again seen on Sept. 23, 1946 for an urticaria of seven weeks' duration. Put on a dosage of 200 mg. of "pyribenzamine" daily, she showed an immediate improvement of symptoms. She was hospitalized on Oct. 10, 1946 for environmental and dietary trials. The etiology of the urticaria was not determined by the initial or subsequent investigations. The patient was discharged on Oct. 29, 1946 with the urticarial outbreaks improved but continuing. At this time examination of her blood revealed a count of 5,700 leukocytes, with a differential of polymorphonuclear neutrophils 74 per cent; lymphocytes 22 per cent, and monocytes 4 per cent. The dose of "pyribenzamine" was reduced to 150 mg. daily, and its administration was continued. Chloral hydrate 0.6 Gm. was given nightly. Senna leaf infusion, self-prepared, completed the list of drugs she was taking at this time, except for nicotinic acid, 100 mg. daily from November 18 to 20. (Subsequently, when the blood count had returned to normal, nicotinic acid was again given without effect on the leukocyte picture.)

Hematologic Data

Date	White Blood Cells Total	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %
4/ 6/42	8,600	55	37	7	1	..
9/23/46	8,000	58	37	..	5	..
10/10/46	5,700	74	22	4	..	..
11/23/46 (11 a.m.)	1,600	12	86	2	..	..
11/23/46 (4 p.m.)	1,300	3	92	5	..	..
11/23/46 (11 p.m.)	1,550	7	83	10	..	..
11/25/46	2,800	48	50	2	..	..
11/27/46	2,600	33	59	8	..	..
11/29/46	2,400	22	71	7	..	..
12/ 1/46	4,400	47	48	4	1	..
12/ 3/46	4,500	52	44	4	..	..
12/ 5/46	6,100	64	30	6	..	..
12/ 9/46	9,200	68	24	8	..	..
12/13/46	8,800	62	30	7	..	1

Hemoglobin 75% (12.8 Gm.); red blood cells 4,400,000; platelets 246,000; reticulocytes 0.6%.

The patient was again hospitalized on Nov. 23, 1946. She had not felt well for a week. An elevation of the temperature to 102 F. had occurred three days before admission. Two days before, a slight, nonproductive cough had developed. On the morning of admission the temperature was 103 F., and bouts of chilliness were experienced. Slight nausea and a dull headache were complained of, but there was no soreness of the throat. Apart from the fever, the physical examination was noncontributory. The chest was clear. No ulceration or inflammation of the mouth or throat was present. A superficial ulcer on the left labium majus, recurrent for years, was healing. Blood culture and routine urinalysis were negative. The blood count was: leukocytes 1,300; polymorphonuclear neutrophils 3 per cent; lymphocytes 92 per cent; monocytes 5 per cent; erythrocytes 4,400,000; hemoglobin 75 per cent (12.8 Gm.); platelets

From the Allergy Service, Medical College of Virginia.

246,000; reticulocytes 0.6 per cent. "Pyribenzamine" therapy was discontinued immediately. Treatment with penicillin, 50,000 units intramuscularly every three hours, was instituted on admission. This was reduced next day to a dosage of 25,000 units every three hours until November 29, at which time it was discontinued. The temperature returned to normal the day after admission and remained so. The attendant symptoms also responded immediately. Chloral hydrate was continued in doses of 0.6 Gm. four times a day until November 29. Senna leaf infusion was given nightly throughout the hospital stay. Twelve cubic centimeters of a concentrated liver extract intramuscularly, with 40 mg. of folic acid by mouth, were given daily from November 30 to December 9. Slight, occasional outbreaks of urticaria were treated by the injection of a solution of epinephrine subcutaneously.

Scratch tests to "pyribenzamine" in solution were negative. Following the method of Leftwich,[1] direct intradermal tests with serum from another patient being given the drug were negative. Attempts were made to demonstrate leukocyte sensitivity to "pyribenzamine" by in vitro lysis of leukocytes by the method of Squier and Lee.[2] A 0.01 per cent solution of the drug in isotonic solution of sodium chloride was added in equal parts to the heparinized blood of the patient and to controls. Serial white counts at intervals during two hours were suggestive, but not conclusive, of the destructive action of "pyribenzamine" on the patient's leukocytes in vitro. It was considered inadvisable to test the patient directly by the readministration of the drug.

SUMMARY

Fever and a marked depression of granulocytes in the peripheral blood, developed in a woman aged 73 after eight weeks of tripelennamine hydrochloride, "pyribenzamine" (N. N. R.), therapy in moderate doses for chronic urticaria. After withdrawal of the drug and the intramuscular use of penicillin, the temperature dropped to normal in twelve hours. The total leukocyte count and the differential percentage returned to normal in twelve days. The neutropenia is thought to have been the result of the drug.

Council on Foods and Nutrition

PLAN FOR ACCEPTANCE OF JUICES

In order to encourage the production of fruit juices containing high natural levels of vitamin C, the Council accepts orange juice in which at least 40 mg. of ascorbic acid per hundred cubic centimeters of juice is conserved.

The following product meets the standards and rules established by the Council for fruit juices.

Attention is called to the fact that this product, while packed in a hermetically sealed container, is not heat treated and should be consumed promptly after defrosting. If it is not to be used immediately after defrosting, it may be kept under refrigeration of approximately 35 degrees F. in the unopened container for three or four days. JAMES R. WILSON, M.D., *Secretary.*

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Calories.—0.55 per gram; 15.62 per ounce.

Vitamin Content.—	Mg. per 100 Cc.
Ascorbic acid	
In juice	43.2
In fruit (before processing)	43.8

1. Leftwich, W. B.: Intradermal Test for Recognition of Hypersensitivity to Sulfonamide Drugs, Bull. Johns Hopkins Hosp. 74: 26-48, 1944.

2. Squier, T. L., and Lee, H. J.: Lysis in Vitro of Sensitized Leucocytes by Ragweed Antigen, read at the meeting of the American Academy of Allergy, New York, Nov. 26, 1946.

Council on Physical Medicine

REPORT OF THE COUNCIL

The Council on Physical Medicine has authorized publication of the following report.

The Council wishes to express its appreciation for the valuable cooperation of the Consultants on Electrocardiographs in reviewing this report. The consultants are Drs. A. R. Barnes, George Fahr, Harold E. B. Pardee, William D. Stroud, Carl J. Wiggers and Frank N. Wilson. HOWARD A. CARTER, *Secretary.*

MINIMUM REQUIREMENTS FOR ACCEPTABLE ELECTROCARDIOGRAPHS

1. The electrocardiographs shall be equipped with a suitable recording mechanism.

2. The recorded response of the instrument to externally applied square wave voltages shall be adjustable to a sensitivity of 1 cm. per millivolt when this voltage is applied to the leads of the instrument through a series resistance of 2,000 ohms. This sensitivity shall be maintained without further adjustment within ± 5 per cent for a period of thirty minutes under operating conditions. Operating conditions for the purpose of this requirement are defined as (1) for alternating current operated instruments as line voltages varying from 105 to 130 volts at frequencies within ± 2 per cent of the specified value for the instrument, (2) for battery operation as one or more of the batteries operating at 80 per cent of the rated life or voltage whichever condition occurs first. Under these conditions the response of the instrument to its incorporated standardizing signal of 1 millivolt shall be within ± 5 per cent of the response to the externally applied test signal. The instrument shall incorporate means of superimposing its intrinsic test signal into the cardiographic tracing as recorded from any lead position. It should be possible to maintain this test signal voltage for a period of two or more seconds.

3. The amplitude response of the instrument to 1 millivolt peak sinusoidal voltage variation up to 15 cycles per second shall not fall below 90 per cent, and up to 40 cycles shall not fall below 50 per cent of the square wave response to equivalent voltage variations. The amplitude response of the instrument to 1 millivolt peak sinusoidal voltage variation up to 300 cycles per second shall not exceed 100 per cent of the square response to equivalent voltage variations.

4. The response of the instrument at 0.2 second after the application of a direct current of 1.0 millivolt shall not deviate more than ± 10 per cent from the response at 0.04 second. The test voltage of 1 millivolt should be applied to the leads of the instrument through a series resistance of 2,000 ohms.

5. When the instrument is adjusted to the sensitivity specified in requirement 2 the recorded response shall be directly proportional to the applied voltage (direct current) within ± 5 per cent over a range of 2 cm. on either side of zero.

6. With the two input terminals connected together, a potential difference applied between them and ground should not produce a deflection of more than 1 per cent of that produced by the same potential difference applied between the two input terminals.

7. The instrument shall incorporate a means of continuously recording time intervals on the record. These intervals shall be of one second duration or less and shall be accurate within ± 2 per cent. A means of superimposing this time signal on the electrocardiographic tracing at the operator's will will be accepted as fulfilling this requirement. It should be possible to record the time signal for a period of at least two seconds. Recording paper preruled so as to indicate time intervals, assuming a constant paper speed, shall not be construed as fulfilling this requirement.

8. The construction of the instrument shall be such as to meet the shock hazard requirements of the Underwriters' Laboratories, Inc.

9. Instruments shall be submitted and considered according to the Official Rules of the Council on Physical Medicine.

10. The standards of merchandising and the acceptability of advertising shall meet the Rules of the Council on Physical Medicine.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

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SATURDAY, MAY 31, 1947

CENTENNIAL SESSION

As The Journal goes to press with this Centennial Number, arrangements have been completed for the largest and most comprehensive session of the American Medical Association ever held. Over 10,000 physicians have already indicated their intention to be present. Rooms have been assigned to all of those who have applied. The Local Committee on Arrangements in Atlantic City, headed by Dr. David B. Allman, announces that additional rooms are available in excellent hotels in the suburbs of Atlantic City. Regular bus service will be provided without charge from these hotels to the Boardwalk. Any one who wishes to attend the annual session should not hesitate to come because of lack of suitable accommodations. The hotel committee, under Dr. Robert A. Bradley, 16 Central Pier, Atlantic City, N. J., will do everything possible to secure satisfactory accommodations for any Fellow of the American Medical Association who wishes to participate in this notable event.

Elsewhere in this issue of The Journal are examples of telegrams, letters, resolutions and editorials that have been published in various periodicals and sent directly to the headquarters of the Association, some in connection with the actual birth date of the Association, May 5. Information is available that many periodicals with large national circulation will also have special issues to commemorate the occasion.

National broadcasts are scheduled for practically every day of the week beginning Sunday, June 8, at which time the speakers at the religious service, Monsignor Fulton J. Sheen, Dr. Joshua Loth Liebman and Dr. Ralph Cooper Hutchison, will be presented at 11 a. m., Eastern daylight saving time.

The Journal has noted previously the issuing by the Post Office Department of a stamp commemorative of the Centennial. The third assistant postmaster general, Mr. Joseph Lawler, is coming to Atlantic City in person to present a sheet of the stamps to the House of Delegates. The J. B. Lippincott Company has arranged to issue a special envelop with a cachet or device commemorative of the occasion and carrying one of the new stamps, so that every physician in attendance may have some to mail to his home.

"The History of the American Medical Association" is in the bindery, and the W. B. Saunders Company, which is the publisher, announces that the book will be available in Atlantic City.

The exhibits, including both the Scientific and the Technical, are most comprehensive, and practically all exhibitors have entered into the spirit of the occasion. Many of these exhibits will be devoted to the history of the advancement of medical science. Others will include mementos of the Centennial for the physicians who visit the exhibits.

The distinguished foreign guests constitute an amazing list representative of medicine throughout the world. Other notables in attendance will include the surgeons general of the various services, Secretary of War Robert P. Patterson, Assistant Secretary of the Navy for Air John Nicholas Brown and possibly also the Secretary of the Interior, Julius A. Krug.

Attention should be called again to the fact that the opening general meeting will be held in the large auditorium on Tuesday evening, June 10. The Philadelphia Festival Orchestra under Alexander Hilsberg will give a concert, which will begin at 7:45. The meeting itself will begin at 9 o'clock. Seated on the stage will be the officers and the Past Presidents of the American Medical Association, the distinguished representatives of the Medical Society of New Jersey and of Atlantic County, and the foreign guests who will be presented on this occasion. This meeting is open both to the medical profession and to the public. Arrangements can be made for seating more than 5,000 people. However, Fellows of the Association who wish to attend should arrive not later than 7:30 so that all may be properly seated before the concert begins.

Two excellent orchestras, those of Tommy Tucker and Eddie Lane, have been secured to supply the music for the President's Reception on Thursday night, June 12. Because of the number of distinguished persons in attendance, there will be three receiving lines. The reception will begin promptly at 9 o'clock. This will also be held in the large hall of the Atlantic City auditorium.

The scientific program for this session of the Association has been especially designed to commemorate one hundred years of medical progress. Each of the sections has a special contribution by a leader in the specialty concerned, and there is to be a special session on the history of medicine.

The celebration of the Centennial has been carefully planned and has required attention to detail beyond any similar occasion in the history of the Association. Every physician who can possibly be present should

endeavor to come to the session, if not for the entire week, at least for one day, in order that he may look back in the years to come with pride of personal participation in this celebration.

DIETARY TREATMENT OF HYPERTENSIVE DISEASE

Shortly after the beginning of the twentieth century Achard and Loeper [1] and Widal and Lemierre [2] demonstrated that a diet of low salt content was helpful in hypertensive disease. Allen in 1920 also proposed a salt free diet for hypertension, but his studies and conclusions did not receive general acceptance by the medical profession.

The possibilities of treating hypertensive disease and its complications by dietary methods has become the subject of renewed study. Among such investigations are those of Grollman and his colleagues,[3] Selye,[4] Wheeler and his co-workers [5] and Kempner.[6] Grollman and his co-workers found that the blood pressure of rats with experimental renal hypertension could be lowered by administering diets in which the sodium content had been decreased by dialysis. Only 6 cases were reported, the reduction of pressure being to normal levels in 2, moderate reduction in 3 and no decline at all in 1. The radical reduction of sodium in the food employed by these investigators cannot be readily duplicated.

Selye, working with experimental animals, found that low sodium and low protein diets would prevent the nephrosclerosis and hypertension which could be produced in these animals by corticotrophen, desoxycorticosterone and other salt active corticoid hormones. In a small number of human beings with rapidly progressive hypertensive disease similar dietary measures were considered effective. Selye believes that the low content of protein and sodium in some of the diets which have been empirically proposed for the treatment of hypertensive disease is the basis of the resultant favorable reports, such as the so-called rice diet for hypertension studied by Kempner.

The volume of experimental and clinical studies and the scientific standards of many of them would seem to confirm the view that a diet low in sodium is helpful for a high percentage of patients with hypertensive disease. Indeed, Wheeler and his co-workers believe that the use of a diet low in sodium in the presence of congestive failure of the heart is also based on sound experimental and clinical grounds. Not all patients of this type will respond. These investigators found that patients with congestive failure due to coronary or hypertensive heart disease responded much better than those in whom rheumatic heart disease was the cause of the failure.

Further work on this therapeutic approach is needed. In spite of the overwhelming evidence of value of a special dietary regimen, agreement on specific constituents of the diet and the indications and contraindications has not been obtained. Careful clinical observations under controlled conditions are necessary before the dietary regimen can be considered worthy of general adoption in clinical practice.

1. Achard, C., and Loeper, M.: Sur la rétention des chlorures dans les tissus au cours de certains états morbides, Compt. rend. Soc. de biol. **53**: 346, 1901.
2. Widal and Lemierre: Pathogénie de certains oedèmes brightiques: Action du chlorure de sodium ingéré, Bull. et mém. Soc. méd. d. hôp. de Paris **20**: 785, 1903.
3. Grollman, Arthur, and others: Sodium Restriction in the Diet for Hypertension, J. A. M. A. **129**: 533 (Oct. 20) 1945.
4. Selye, Hans: The General Adaptation Syndrome and the Diseases of Adaptation, J. Clin. Endocrinol. **6**: 117 (Feb.) 1946.
5. Wheeler, Edwin O., and others: Diet Low in Salt (Sodium) in Congestive Heart Failure, J. A. M. A. **133**: 16 (Jan. 4) 1947.
6. Kempner, Walter: Treatment of Kidney Disease and Hypertensive Vascular Disease with Rice Diet, North Carolina M. J. **5**: 125 (April) 1944; Treatment of Kidney Disease and Hypertensive Vascular Disease with Rice Diet: II, ibid. **5**: 273 (July) 1944.

DIAGNOSTIC MEDICAL CARE AND COMMERCIAL LABORATORIES

Accurate diagnosis is the cornerstone of modern medical care. Following adequate review of the patient's history and complete physical examination supplemental clinical laboratory and x-ray examinations may be necessary in many cases. The physician determines, following these additional diagnostic studies, whether or not still further tests, such as functional and neuropsychiatric tests, bronchoscopy and gastroscopy, are required in a much smaller number of cases. Diagnostic medical examinations which involve pathologic and radiologic investigations and clinical laboratory studies may be done in doctors' offices and in hospitals. Fundamental to any examination is the availability of trained personnel that it may be conducted properly and interpreted adequately. In the larger centers, radiologists and pathologists (qualified physicians) usually conduct these examinations. In smaller communities, circumstances often require that they be performed by general practitioners who have had some supplemental training in radiology or pathology.

The profound importance of competent pathologic examination is evidenced in the early diagnosis of malignant disease. Consider the woman with a lump in her breast or the youth with a painful lesion involving bone. In either case carcinoma may be present. If it is, the swiftest method of cure is removal of the breast or leg. On the decision of the pathologist in most instances this grave step must be taken. If there is a well trained pathologist accessible to the hospital or physician's office the patient is likely to receive a correct diagnosis so that a life saving measure is then justifiably undertaken. If, however, reliance is placed on a poorly trained or unqualified person needless and mutilating operations may be performed for benign tumors. If a diagnostic procedure such as a simple blood count or a roentgenographic study of the chest is performed by competent personnel under the supervision of and responsible to a specialist in one of those fields, the

end result is likely to be earlier diagnosis and better medicine. An incompetent, untrustworthy test is often worse than none!

In the early days of clinical laboratory and roentgenologic medicine technicians (notably after World War I) sometimes offered their services as practitioners in these fields. Many were moderately experienced. Owing to the shortage of trained medical specialists, these technicians were frequently accepted and supported by the medical profession. In the meantime, departments of radiology and clinical pathology were becoming established in medical schools, and young physicians were being encouraged by the faculties to enter these fields. Today the continued development of such competent specialists may well depend on the extent to which commercial laboratories, supervised, owned or controlled by nonmedical personnel, are eliminated from the field.

In some parts of the country the commercial laboratory has become synonymous with convenient diagnoses and even rebates. To the credit of the medical profession such practices are exceptional. Nevertheless the signs are clear. Physicians must support medical laboratories which meet the standards of the Council on Medical Education and Hospitals, preferably owned and conducted by medical men trained in their fields. There should be no place in medicine for commercial nonmedical interests that exploit both patient and physician and undertake without recognized responsibility decisions that can rest safely only on those licensed to practice medicine and specially qualified in their fields.

Current Comment

LATE RESIDUALS OF INFECTIOUS HEPATITIS

An estimated 90 to 95 per cent of patients with acute infectious hepatitis appear to recover entirely within a period of two to three months. Klatskin and Rappaport [1] have attempted to determine the incidence of residuals in a large group of patients presumably recovered from this disease. The studied group consisted of 217 persons presumably cured of infectious hepatitis, of whom 47 had symptoms, 59 had an enlarged liver and 41 had evidence of impaired liver function—a total of 108, eliminating duplications, or about 50 per cent of the cases. The significant symptoms were right upper quadrant pain and fat intolerance. Structural changes were not established, though the evidence suggests that the residuals mentioned indicate a disturbance of the liver. Good health and full activity over long periods—the time varying from two months to twenty-seven years—were compatible with these changes. The severity of the jaundice at the time of the disease was the only symptom which appeared to play a significant part in the incidence of residual changes. Evidence could not be deduced from the patients studied that the residuals described predispose the liver to further injury. Likewise the presence of late residuals of infectious hepatitis did not appear to reflect any tendency to develop cirrhosis, though coarse nodular cirrhosis occasionally occurs during the first six months of chronic progressive or recurrent infectious hepatitis.

1. Klatskin, Gerald, and Rappaport, Emanual M.: Late Residuals in Presumably Cured Acute Infectious Hepatitis, Ann. Int. Med. **26**: 13 (Jan.) 1947.

TENNESSEE AND THE NATUROPATHS

In 1943 and 1945 the general assembly of Tennessee passed a law legalizing the licensing of naturopaths. A naturopathic board was established to deal with such licensure. Now by an act of the last general assembly in Tennessee the naturopathic board has become extinct. Within a week after the law was first passed a member of the naturopathic board sent a letter to naturopathic healers through the nation telling them that Tennessee had now become the happy hunting ground for their profession. The publication of the Tennessee Department of Public Health states that a horde of naturopathic healers met at the Maxwell House in Nashville four months later and hundreds of licenses were issued like tickets to a banquet. A reporter for the *Commercial Appeal* estimated that about 1,500 Tennessee licenses to practice naturopathy were doled out to those able to purchase them; evidence was developed indicating the establishment of a political organization for the maintenance of this unfortunate system of practice. Among other naturopaths who migrated to Tennessee was William E. Estep, who has a long list of bogus performances and fake licenses to his credit. The Memphis newspaper gives an amazing account of a visit by one of its reporters, Harry Woodbury, to Estep's clinic. When the reporter entered he was greeted by a man sitting behind a plain desk who said to him "You look like you are in quite a bit of pain. What seems to be troubling you, sir?" The reporter submitted to an examination by Estep during which the naturopath said to him "You are eaten up with sinus!" He was also told by Estep that he had stomach ulcers and that his blood pressure was dangerously high. A previous examination had shown that the reporter was not actually subject to any of these maladies. The naturopath gave Mr. Woodbury an electrical treatment and three prescriptions, one of which called for 4 ounces of sulfocyanate. Estep was indicted for practicing medicine without a license and sentenced to a fine of $500 and imprisonment in the county workhouse for eleven months and twenty-nine days. The Tennessee incident is reminiscent of the diploma and license scandals of a previous generation. It calls to mind the shipping of carloads of physicians with well nigh worthless diplomas from schools in St. Louis and Kansas City to Connecticut and Arkansas, where conveniently eclectic boards licensed them. That was the scandal that gave the United States such notorious charlatans as Brinkley and Schireson. Who knows how long Tennessee and the rest of the United States will be paying for the folly of the general assemblies of 1943 and 1945?

ORGANIZATION SECTION

CONGRATULATIONS AND GREETINGS

Note.—Here are examples of congratulatory messages, editorials and letters received in the headquarters office in association with the Centennial of the Association.—Ed.

A. M. A. FOUNDERS

(The Evening Star, Washington, D. C., May 13, 1947)

Emerson's doctrine that a great institution is "the lengthened shadow of one man" is subject to amendment in certain cases. The American Medical Association, now celebrating the centennial of its establishment, was the result of a matching of the minds of a number of men of distinction. It grew from two conventions, held at New York and at Philadelphia in May 1846 and May 1847. The constitution in its current form declares: "The object of this Association shall be to promote the science and art of medicine. Contributing to this end, the association shall endeavor to unite into one compact organization the medical profession of the United States for the purpose of fostering the growth and diffusion of medical knowledge, of promoting friendly intercourse among American physicians, of safeguarding the material interests of the medical profession, of elevating the standard of medical education, of securing the enactment and enforcement of just medical laws, of enlightening and directing public opinion in regard to the broad problems of hygiene and of representing to the world the practical accomplishment of scientific medicine."

Foremost among the primary sponsors of the ideals thus set forth was Dr. Nathan Smith Davis, who as early as 1843 proposed such a movement. His major interest was in the better training of medical practitioners—a concern which was shared by the celebrated psychiatrist Dr. Pliny Earle and by the chairman of both of the preliminary conventions, Dr. Jonathan Knight. All three of these founders were possessed of a genius for persuading people of different views to work together.

Another generation than that of the original organizers produced Dr. Nathaniel McCormack of Kentucky, who, with the assistance of several colleagues, revised the constitution of the Association and drafted the prevailing plan for the management and conduct of its affairs. He too deserves to be remembered in connection with the centenary celebration.

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MEDICAL CENTENARY

(New York Times, May 7, 1947)

On May 5, 1847 some 250 delegates representing more than forty medical societies and twenty-eight colleges met at Philadelphia to form what has ever since been the American Medical Association. A century later the Association has 131,590 members and 72,243 Fellows. No other body wields greater professional influence. During the long decades when the government was indifferent to the quality of food and drugs and the "patent" medicine man was in his heyday, the Association was a bulwark of protection. Quacks were pursued, educational standards were raised, drugs were appraised, hospitals and schools were approved or disapproved, a dozen first class medical publications, of which THE JOURNAL is the best known, were made indispensable purveyors of knowledge, nostrums were exposed as the frauds that they were, fads were riddled.

ANOTHER FELLOW WHO STARTED OUT A HUNDRED YEARS AGO

Published in the Chicago Daily *Tribune*, May 7, 1947. The *Tribune* also is celebrating its centennial in June.

For all these blessings the public has reason to be grateful. With some of the economic and social policies of the Association we have disagreed. But certainly in intelligence, in education, in breadth of culture the Association is not surpassed by any other organization, because no other so carefully excludes from its ranks the badly trained, the charlatans and the sectarians. We wish it another century of usefulness.

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HOSPITALS AND THE A. M. A.

(Hospital Management, May 1947)

In the leading article the writer states that "hospitals may well share the pride of the Association in the completion of its hundred eventful years, for the history of the American Medical Association, and of the medical profession in general, is irrevocably linked with the history of hospitals. The two have worked as a smooth-running team, complementing and supplementing each other's activities with the same broad objective in mind: the betterment of the health of mankind."

* * * *

CENTENARY OF THE ASSOCIATION

(Good Housekeeping, June 1947)

In Atlantic City in June the American Medical Association will observe its Centenary, and so it is appropriate that we extend to the Association our sincerest congratulations and good wishes. It is doubtful whether any other organization has had as much of an influence for good on American life. Organized medicine in this country is powerful, as it deserves to be and must be. But for the highly centralized headquarters in Chicago we would not now be so free of quacks, and of bad medical legislation, and of medical plans and schemes that take advantage of a gullible public. Immeasurable good has come from the American Medical Association. Largely because of it the status of medicine in America is unexcelled; the stature of the profession is the highest ever known. We salute the Association; and, in honoring it, we honor a group of indispensable citizens.

A. M. A. CENTENNIAL

(Drug Trade News, May 19, 1947)

The news that the American Medical Association reached its one hundredth birthday on May 5 is alone sufficient to spotlight its profoundly valuable contributions to the advancement of medicine and the general welfare.

Whatever the field of medical interest, be it education, hospital development, public health administration, research, expansion of medical care, drug control, journalism, a study of its history will show that the A. M. A. has always been the dominant voice demanding ever higher standards and ever greater public benefits.

From the outset, the A. M. A. became dedicated to the task of progressively improving medical education, and so effectively did it achieve that today medical education in the United States is by all odds vastly superior to that in any other section of the world.

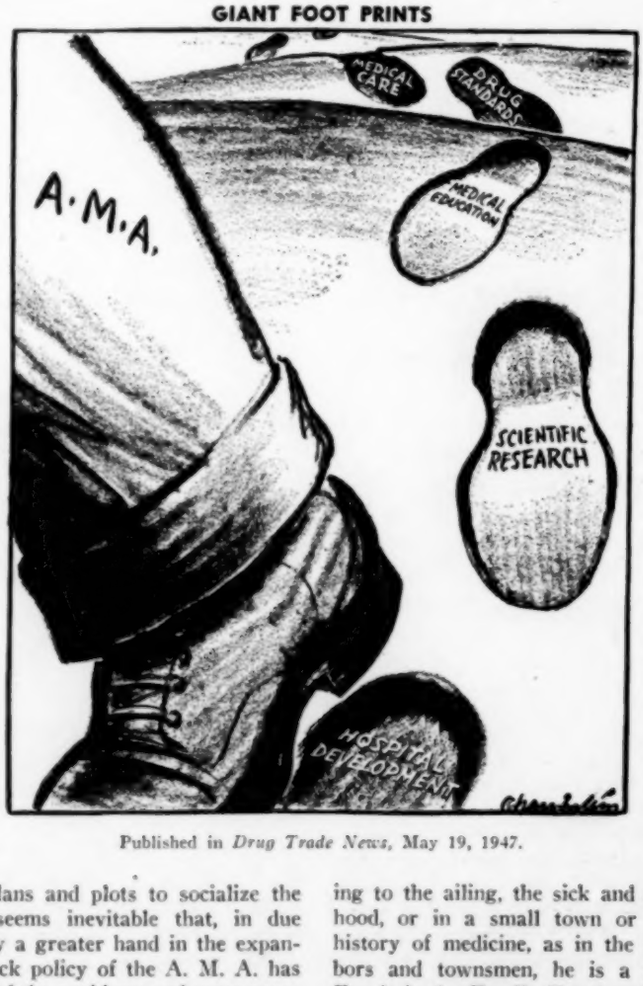
In the field of drug standardization, control and promotion, the A. M. A. has, through its Council on Pharmacy and Chemistry, been a most constructive influence in behalf of greater scientific accuracy and more dependable therapeutic agents. Here its leadership, which necessarily includes the element of discipline, may well be said to have instituted new concepts of pharmaceutical integrity.

The A. M. A. has been the strong, motivating factor underlying medical research. It has fostered the establishment of research departments and laboratories in the medical schools, in various governmental services and in the great pharmaceutical industry plants. The high standards of scientific effort which now obtain in so many areas of medical care are a tribute to the A. M. A. conviction that science and medicine are partners in the cause of human betterment.

Quite properly, we think, the A. M. A. has fought the many half-baked, visionary schemes, plans and plots to socialize the medical profession. While it seems inevitable that, in due course, the government must play a greater hand in the expansion of medical care, the hold-back policy of the A. M. A. has assured a more careful appraisal of the problems and more practical programs for meeting them.

By every test, the A. M. A. has gloriously justified its first hundred years.

GIANT FOOT PRINTS



Published in *Drug Trade News*, May 19, 1947.

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CENTURY OF PROGRESS

(Boston Post, April 30, 1947)

On May 5 the American Medical Association will be 100 years old. It has had a century of growth that is truly phenomenal. Starting with a bare handful of physicians, today its rolls include 131,590 members and 72,243 Fellows. Its accomplishments in the field of public health too have been notable. Always in the forefront in the prodigious advancement of medical science during that period, it well deserves national recognition at this first major milestone for the fine job it has done.

THE DOCTOR

(The Lion, May 1947)

Next month the American Medical Association will celebrate its Centennial, it being just one hundred years ago that that organization, so great in accomplishment and rich in service to humanity, was founded in Philadelphia. At that time the medical profession was still in an exceedingly primitive state, the practitioners being for the most part men with little knowledge of the human body and much less of the cause and nature of disease, and who in treatment relied largely on blood letting, nostrums and superstition. There were no professional standards and no medical ethics. Yet there were here and there a few enlightened physicians and surgeons who recognized their responsibilities and followed the higher ideals of their calling, and it was a group of these men who in 1847 banded together into an association for the primary purpose of elevating medical education, promoting research and establishing the standards and principles of ethics so sorely needed for public protection.

The contrast between medicine as it was studied and practiced in 1847 with medical education and practice of today is so vast as to represent one of the greatest marvels of modern times. During the forthcoming Centennial it is but fitting that special honors will be paid that vast body of physicians, surgeons and specialists in the various branches of medical science who during these hundred years have developed the remarkable diagnosis, treatment and surgical technics that have conquered many of the diseases that formerly scourged mankind, that have brought miraculous cures to untold millions of sufferers and that have increased the span of life by many years.

But the spotlight will specially fall during this Centennial on that kindly friend and benefactor of humanity, the Family Doctor. Maybe he is unknown to the world at large. Maybe he is spending his life in relative obscurity, ministering to the ailing, the sick and distressed of his own neighborhood, or in a small town or rural community. But in the history of medicine, as in the hearts and minds of his neighbors and townsmen, he is a man of greatness nevertheless. For it is the Family Doctor who is the core of his profession. Not only has all medical knowledge sprung from the experiences of his practice, but he is the medium through whom the century's medical progress is made a reality for us all. And, in thinking of the Family Doctor as the consecrated and faithful servant of humanity, we recall the dramatic characterization of him presented by the famous painting "The Doctor," which is reproduced on the front cover of this issue of the *Lion*. It is the masterpiece of Sir Luke Fildes, which hangs in the Tate Gallery in London and which was exhibited in the Hall of Science at the Century of Progress Exposition in Chicago in 1933-1934. Not only is the painting itself dramatic, but the story behind it is equally so.

Nearly sixty years ago Queen Victoria of England arrived at one of her rural estates to find one of her servant women and her husband grief stricken because their only child lay at the point of death. The Queen immediately telegraphed to London

for her personal physician, Sir James Clark. He arrived by special train and remained in constant attendance on the child until the crisis was past and she was well on the way to recovery. The girl was literally snatched from the grip of death, and Queen Victoria was so impressed with this medical miracle that she commanded Sir Luke to place on canvas the scene in the humble cottage as the child's life was feebly flickering, as a tribute to the entire medical profession for its devotion to the cause of humanity.

Lionism prides itself on the many thousands of men of medicine within its ranks, in all of its eighteen countries. Ever since the founding of Lions International three decades ago, medical men have been among the most active Lions Club members. They have been tireless workers not only in their own professions but in every Lions Club activity for the welfare of their city or town and area, and the number of them who have occupied high posts in Lionism is countless.

Lionism also prides itself on the fact that, just as the medical profession has cooperated intensely in the growth and development of our Lions Club and Association, so has it been privileged to cooperate with the medical profession in a great many ways. Lions clubs in all our eighteen countries have sponsored and very often financed the construction of numerous hospitals, clinics and special wards, have equipped them with laboratories, diagnosis and surgical equipment, furnishings and facilities of every kind. They have served communities lacking adequate medical services by locating competent doctors in them, have built blood banks, have assisted in spreading public health education, have promoted modern sanitation—to mention only a few such activities.

Lionism, in a word, could never have been the great constructive force in public service that it is today were it not for this mutual cooperation by and with the men of medicine, and Lions everywhere salute them on this Centennial of their first professional organization.

MELVIN JONES.

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ONE HUNDRED YEARS OF PROGRESS

(Grand Rapids, Mich., Herald, May 10, 1947)

Medical science as it is known today has made its greatest advancements in the last hundred years. It may indeed be said to have been the product of the last century.

And it is more than coincidence that this great period should have been the period of the American Medical Association, founded May 5, 1847 and now celebrating its centennial.

In this country, the A. M. A. has taken the lead in elevating the standards of medical education and practice, in the elimination of "quacks" and "quack" medicines, in promoting research and in improving the public health.

Countless comparisons might be made between the methods and knowledge and standards of today and those of a century ago. They are not necessary. Most of us in our lifetime have seen the results of progress in too many lines of medical practice to need comparisons with the crude ways and sparse facts of a hundred years ago.

One by one, and in large measure due to the organization that now numbers 131,590 of America's 190,000 doctors of medicine in its membership, we have seen the passing of fads and frauds and the introduction of new skills and new drugs.

Nor have the art and science of medicine reached the end of progress. Its practitioners will go on and on, finding new and newer means of alleviating suffering and saving lives and making life more worth living.

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NOTABLE CENTENNIALS

(Northwest Medicine, May 1947)

It should be known by every physician that the Atlantic City meeting of the American Medical Association in June will celebrate its one hundredth anniversary. Whether such an occasion distinguishes an individual or an organization, it is one of the most notable occasions that can be observed. Whatever distinction is thus conveyed to our national medical association, it is merit and honor for the accomplishments of this organization for the benefit of the medical profession of this country and the welfare of its population.

Undoubtedly a record attendance will be recorded at this meeting, and the official registration of the first day will be crowded and confused. Fortunately, if one desires to take advantage of this privilege, one can obtain from Dr. Lull, Secretary of the Association, a questionnaire which may be filled in advance and save the time and trouble of registration at the meeting.

Another notable centennial celebration is that of the New York Academy of Medicine which has served the medical profession and citizens of New York City for one hundred years. It has maintained the highest standard of service with constant accomplishments for the public health, providing medical information as well as instruction. Its greatest activity is through its magnificent reference library in one of the city's outstanding edifices on Fifth Avenue and 101st Street. This is one of the largest and most unusual medical libraries of the nation. The New York Academy of Medicine is to be congratulated for its conspicuous and successful career during the past century.

(TELEGRAMS)

From the Medical and Chirurgical Faculty of Maryland:

THE MEDICAL AND CHIRURGICAL FACULTY OF MARYLAND EXTENDS HEARTY CONGRATULATIONS TO THE AMERICAN MEDICAL ASSOCIATION ON ITS ONE HUNDREDTH BIRTHDAY MAY 5TH 1947 AND BEST WISHES FOR A MOST SUCCESSFUL MEETING ON JUNE NINTH TO THIRTEENTH WHEN YOU TAKE RECOGNITION OF THIS ANNIVERSARY.

W. HOUSTON TOULSON, M.D., SECRETARY.

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From the Tristate Hospital Assembly:

SIX THOUSAND MEMBERS OF THE TRISTATE HOSPITAL ASSEMBLY OPENING THEIR SEVENTEENTH ANNUAL MEETING TODAY AT THE PALMER HOUSE SEND HEARTIEST CONGRATULATIONS TO THE AMERICAN MEDICAL ASSOCIATION ON ITS ONE HUNDREDTH ANNIVERSARY.

MALCOLM T. MACEACHERN, M.D., CHAIRMAN.

* * * *

From the Chicago Convention Bureau:

THE CHICAGO CONVENTION BUREAU EXTENDS ITS CONGRATULATIONS TO THE AMERICAN MEDICAL ASSOCIATION ON ITS ONE HUNDREDTH ANNIVERSARY. YOUR ACCOMPLISHMENTS HAVE SHOWN YOU TO BE ONE OF THE OUTSTANDING ORGANIZATIONS OF THE WORLD AND WE OFFER OUR BEST WISHES FOR YOUR CONTINUED GROWTH AND SUCCESS.

I. S. ANOFF,
PRESIDENT, THE CHICAGO CONVENTION BUREAU.

* * * *

From the American Protestant Hospital Association:

THE AMERICAN PROTESTANT HOSPITAL ASSOCIATION EXTENDS CONGRATULATIONS ON YOUR CENTURY OF SERVICE AND EDUCATION.

ALBERT G. HAHN, EXECUTIVE SECRETARY.

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From Belgium:

BRUXELLES MEDICAL ASSOCIATION SENDS BEST WISHES FOR YOUR CENTENARY.

(LETTERS)

From the Rho Pi Phi Pharmaceutical Fraternity:

Rho Pi Phi Pharmaceutical Fraternity salutes the American Medical Association upon its "century of progress." May the future find the A. M. A. in the front ranks of the march toward the advancement of the Public Health in America.

A Happy and Healthy Birthday.

Very truly yours,
Maurice Goldsmith.

* * * *

From the National Social Welfare Assembly, Inc.:

May I extend the greetings and hearty congratulations of the National Social Welfare Assembly to the American Medical Association and its great membership on the occasion of your Association's one hundredth birthday.

The long and brilliant record of scientific and human care progress in the field of medicine and health that has grown out of the organized interest of the medical profession as expressed in the American Medical Association is one of the gratifying chapters in our American way of doing things. The people of this country owe much to this one hundred years of organized professional achievement.

The voluntary and government organizations affiliated with the National Social Welfare Assembly find many occasions for hearty and close partnership with your members in the furtherance of services of mutual interest. We therefore feel very much a part of your centenary observance. The days ahead will undoubtedly bring even closer associations between the medical profession and organized social welfare, for we are united in the great common purpose of the well being of people.

Sincerely yours,
Robert E. Bondy.

* * * *

From the Editor of the British Medical Journal:

The British Medical Association and the American Medical Association are almost contemporaries, there being only the small gap of fifteen years between the foundation of these two powerful professional organizations.

The American Medical Association is the force it is today because it was built on sound foundations. When the founders met in 1847 in Philadelphia, where the framers of the Constitution had met sixty years before, their first concern was with medical education and medical ethics. The American Medical Association was based on first principles, and during the hundred years that have elapsed everything else has been added to it.

English medicine may be proud that it played a part in your formation and development. Dr. Thomas Percival, F.R.S.—as Dr. Morris Fishbein points out in his "History of the American Medical Association"—formulated his principles of medical ethics, and these served as a model for the American medical profession.

The British Medical Association may boast too that it has shared in what is perhaps your most important development—the establishment of The Journal of the American Medical Association. It is many decades ago now that your Association invited to the United States Dr. Ernest Hart, then Editor of the *British Medical Journal*, to discuss plans for starting a similar journal, similarly linked with a voluntary association of medical men. And we may be proud too that the second Editor of The Journal was an Englishman by birth and an American by adoption—Dr. George H. Simmons, who did such pioneer work both for The Journal and for your Association.

The links between our two associations are close indeed. We look with admiration at the influence exerted by the American Medican Association on all aspects of professional life in the United States. As an editor I should like to pay my humble tribute to the magnificent standard of medical journalism set and maintained by The Journal and by the specialist journals published by the American Medical Association.

In a hundred years the American Medical Association has become what a contemporary medical historian rightly describes as "the largest and most powerful medical organization that the world has yet seen." On this centenary occasion we salute the memory of its founder, Nathan Smith Davis, and wish the Association *bon voyage* for its second hundred years.

Hugh Clegg, M.A., M.B., F.R.C.P.

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From the Philippine Medical Association:

Whereas, on June 9-13, 1947 the great American Medical Association will celebrate the 100th anniversary of its foundation;

Whereas, this great medical association has, during the century of its existence, been consistently useful and progressive;

Whereas, all the wonderful advances in medical education and in public health and sanitation in the great North American nation have been brought about largely through the patriotic and unselfish labors of this medical association with its important state ramifications; and most, if not all, of the medical legislations in the American continent have been made possible only through the harmonious collaboration between this association and the American government;

Whereas, the Philippines—from the first years of its association with the United States, throughout its existence as a Commonwealth, until now that it is a Republic—has, through the Philippine Islands Medical Association, which was at one time accredited as one of the state branches of the American Medical Association, been the grateful recipient of the many benefits of this association; and

Whereas, the American Medical Association, in its one hundred years of useful life, has consistently and painstakingly upheld and guarded the best physical and medical interests of the American people as well as those of the American medical profession in general; therefore be it

Resolved, That the Philippine Medical Association, conscious of its national debt of gratitude to the American Medical Association, hereby expresses its sincere congratulations to the American Medical Association on this auspicious date and conveys the best wishes of the medical profession of the Philippines for the continuance of the useful and vigorous life of its former mother, the American Medical Association.

Januario Estrada,
President.

* * * *

From an address by Alphonse M. Schwitalla, S. J., Dean, St. Louis University School of Medicine, at annual banquet of the Illinois State Medical Society, May 13, 1947:

We are this year celebrating the hundredth anniversary of the foundation of the American Medical Association. It has been a century of leadership by medicine; leadership through the science of medicine; leadership through the practice of medicine, through the ethics of medicine, through medical education, through the thousand and one applications of medical thought to industry and commerce, to government and finance. It has been a century of challenged leadership because many were those who would not acquiesce in the leadership of medicine and

who tried to place their own puny judgments of what is good for mankind in opposition to the combined judgment of medicine as personified in the reputable physicians who stood for competence and medical wisdom. No, I would not exaggerate; the leadership of medicine has not been perfect. There have been moments in a hundred years when lesser councils prevailed, when the bayleaves and olive branches of the first caduceus became the paired serpents and the wizard wand of the second caduceus; when in moments, that were fortunately very rare, the pressure of public opinion at times prevailed over principle; but all in all the triumph is really this, that after one hundred years of effort in medicine man can really look to medicine in America for leadership and find ever so much to attract his loyalty and his allegiance, to elicit his confidence, to inflame his enthusiasm, to command his acquiescent obedience. For medicine in America is a movement besides being a science and an art. It is a philosophy; it is public opinion; it is an enterprise; it is a national activity; it is a dynamic and an essential element in the American pattern; it is a pervasive phase of American psychology and sociology; it is a trend toward the truth, an urge toward morality. All of this man has found in medicine when he looks to medicine.

* * * *

RESOLUTION PASSED BY THE SENATE OF PENNSYLVANIA

On May 5, the American Medical Association will celebrate its one-hundredth anniversary, having been founded in Philadelphia on May 5, 1847.

It was founded as an association of physicians "to promote the science and art of medicine; to elevate the standard of medical education and practice; to bring about the enactment of uniform legislation for the public welfare and to protect the public health."

In connection with its purpose to protect the public health, it has over the years helped initiate many legal actions and has helped to bring innumerable criminal prosecutions against quacks and quackery. The Association has also found itself involved as a defendant in many libel suits because where it has seen medical charlatans and others involved in introducing pseudo medical skullduggery into the field of medicine it has not hesitated to inform the public accordingly and to take those steps which it regarded as the quickest way to stop such practices.

The American Medical Association has during its many years been a constructive influence because of the caliber of the men who have been its officers and members; therefore be it

Resolved, That the Senate of the Commonwealth of Pennsylvania hereby wishes to join in commemorating the centennial celebration of the American Medical Association and to congratulate its officers and members and to extend to them its best wishes in carrying out its many plans for advancing the study and practice of medicine and for protecting our citizens from ill considered medical experiments and questionable drugs.

The Senate also expresses the hope that the American Medical Association at its centennial convention, to be held June 9th to 13th, will give great consideration to the problem of improving the general health of the average American citizen, mindful of the low standard of health which was discovered by our military authorities during the last war; and be it further

Resolved, That the Secretary of the Senate transmit a copy of this resolution to the President of the American Medical Association.

G. Harold Watkins, Secretary
Senate of Pennsylvania.

CENTENNIAL BROADCASTS FROM ATLANTIC CITY

The Bureau of Health Education announces the following broadcasts connected with the Centennial Celebration of the American Medical Association at Atlantic City:

Saturday, June 7. "Doctors—Then and Now," Nevada and Northern California, speaker Lawrence Parsons, M.D., representing Nevada and California Medical Associations; N. B. C. and affiliated stations, 4:30 to 5 p. m., Eastern Daylight Time.

Saturday, June 7. "Generations of Doctors" (tentative title), a fantasy story of the doctor of 1847 in contrast with his descendant, a doctor of 1947, dramatized with music; Columbia Broadcasting System and affiliated stations, 10:30 to 11 p. m., Eastern Daylight Time.

Sunday, June 8. Speakers from the Centennial religious observance, Msgr. Fulton J. Sheen, Rabbi Joshua Loth Liebman and Dr. Ralph C. Hutchison, President of Lafayette College; N. B. C. and affiliated stations, 11 to 11:30 a. m., Eastern Daylight Time.

Monday, June 9. Official opening of the Scientific Exhibit, Dr. Morris Fishbein, Editor of The Journal, and Dr. Harrison H. Shoulders, President of the American Medical Association; American Broadcasting System and affiliated stations, 4:15 to 4:30 p. m., Eastern Daylight Time.

Monday, June 9. "Stephen Graham, Family Doctor": this Mutual network broadcast, usually originating from New York, will be originated from the American Room at the Hotel Traymore as the entertainment feature for the annual dinner tendered the House of Delegates and officers of the American Medical Association by the local medical profession. The entire production will be moved from New York as a demonstration to the delegates and as a special event commemorating the Centennial of the American Medical Association. The topic will be "Medical Service in Rural Areas." The special script will be written by Willys Cooper and produced by Wynn Wright. The program is under the general supervision of Dr. W. W. Bauer and Harriet Hester.

Wednesday, June 11. Address by Dr. Edward L. Bortz: Columbia Broadcasting System, 6:15 to 6:30 p. m., Eastern Daylight Time. Topic: "Looking Forward from A. M. A. Centennial."

Friday, June 13.—Interview: Dr. Edward L. Bortz, President of the American Medical Association; interviewed by Harriet Hester, Mutual Broadcasting System and affiliated stations, 10:30 to 10:45 a. m., Eastern Daylight Time. Topic: "One Hundred Years of American Medicine."

Saturday, June 14. "Doctors—Then and Now"; closing program of the series entitled "American Medical Cavalcade," summarized by Dr. W. W. Bauer; N. B. C. and affiliated stations, 4:30 to 5 p. m., Eastern Daylight Time.

In addition to the Monday night Mutual broadcast of the "Stephen Graham, Family Doctor" show, the speeches by Dr. Fishbein and Shoulders on the American Broadcasting System, and the speeches of Dr. Bortz on the Columbia Broadcasting System and the Mutual Broadcasting System will originate from Atlantic City; "Doctors—Then and Now" on N. B. C. will originate both Saturdays from Chicago; "Generations of Doctors" on C. B. S. will originate from New York.

DISTINGUISHED GUESTS AND VISITORS

Dr. Hugh A. Clegg

Dr. Sven Forssman

Dr. John A. Pridham

Dr. Jose Valls

The accompanying pictures of guests from other countries to the Centennial Session are in addition to those published in THE JOURNAL, May 3.

Dr. Hugh Anthony Clegg, editor of the *British Medical Journal;* general supervisor of the World Abstracting Service, which the British Medical Association started in January 1947; formerly house physician at St. Bartholomew's Hospital, London; assistant medical officer, London County Council; medical registrar, Charing Cross Hospital; author of "What Is Osteopathy?" (jointly), Wartime Health and Democracy; Medicine in Britain (British Council); revised Black's Medical Dictionary. Dr. Clegg will attend the Centennial Session as a representative of the British Medical Association.

Dr. Sven Forssman, professor and head of the department of industrial hygiene, National Institute of Public Health, Stockholm, Sweden; secretary of the Swedish Medical Society; editor of the *Scandinavian Journal of Hygiene;* vice chairman of the Swedish Association of Industrial Physicians; will address the Section on Preventive and Industrial Medicine and Public Health at the Centennial Session.

Dr. John A. Pridham, London, England, chairman, International Relations Committee of the British Medical Association; member of the Council and Insurance Acts Committee; member of the Royal College of Surgeons and of the Organization Committee of the World Medical Association; justice of the peace for Weymouth; author of "Problem of the Care of the Neurotic Patient."

Dr. Jose Valls, Buenos Aires, Argentina, professor of orthopedic surgery, University of Buenos Aires Medical School; president of the Argentina Medical Association and formerly president of the Argentina Society of Orthopedic Surgery; vice president, Section on Orthopedic Surgery, fourth congress of the Pan American Medical Association in 1933 and secretary of the eighth congress, in 1940; chief of orthopedic and traumatic surgery at the Durand Hospital; author of "Low Back Pain and Sciatica"; will address the Section on Orthopedic Surgery at the Centennial Session on "Aspiration Biopsy in the Diagnosis of the Lesions of the Vertebral Bodies."

Dr. Charles Hill, formerly deputy secretary and now secretary of the British Medical Association; formerly clinical assistant, Children's Department, London Hospital; London University Extension lecturer in biology, and formerly chairman, Central Council for Health Education; author of "What Is Osteopathy?" (jointly) and "Wise Eating in Wartime," will attend the Centennial Session as a representative of the British Medical Association.

Dr. Frank S. Patch, Montreal, Canada, for many years professor of urology and chairman of the departments of urology and surgery at McGill University; formerly president of the Canadian Medical Association and managing editor of its journal; formerly president of the Royal College of Physicians and Surgeons of Canada; president of the Montreal-Medical Chirurgical Society; colonel in the Canadian Army Medical Corps in World War I; author of numerous articles in the field of urology; will address the Section on Urology at the Centennial Session.

Dr. Andrew R. Hunter of the Manchester Royal Infirmary, Manchester, England; visiting anesthetist to the Thoracic Unit, Baguley Emergency Hospital; consultant anesthetist to Stepping Hill Hospital, Stockport; special anesthetist in the recent war in the Emergency Medical Service in Scotland; will address the Section on Anesthesiology at the Centennial Session.

Dr. Harry Guy Dain, London, England, chairman of the council of the British Medical Association and of the negotiating committee of the medical profession concerned with national health service legislation; formerly the chairman of the Insurance Acts Committee and of the Representative Body of the B. M. A., will attend the Centennial Session as a representative of the British Medical Association.

Dr. Charles Hill

Dr. Frank S. Patch

Dr. Andrew R. Hunter

Dr. Harry Guy Dain

NEW STAMP TO HONOR AMERICAN MEDICAL ASSOCIATION

(Published in The Columbus [Ohio] Citizen, April 13, 1947)

The United States Post Office Department will issue another commemorative stamp, honoring the doctors of America, it was announced this week by Postmaster General Robert E. Hannegan.

The special stamp will be of the 3 cent denomination and will be placed on sale June 9, on the occasion of the one hundredth anniversary of the founding of the American Medical Association.

"In so honoring the American doctor," Mr. Hannegan said, "we are paying tribute to the men and women of medicine who devote their lives to the cause of humanity.

"The alleviation of pain and suffering and the betterment of mankind are their creed. The contribution which they have made to our national life is one of which all Americans can be proud and grateful."

Details as to the place of sale and a description of the stamp will be announced later, the department said.

J. CHARLES BAXTER.

Bureau of Information

LOCATION OF PHYSICIANS

Virginia Shuler
Chicago

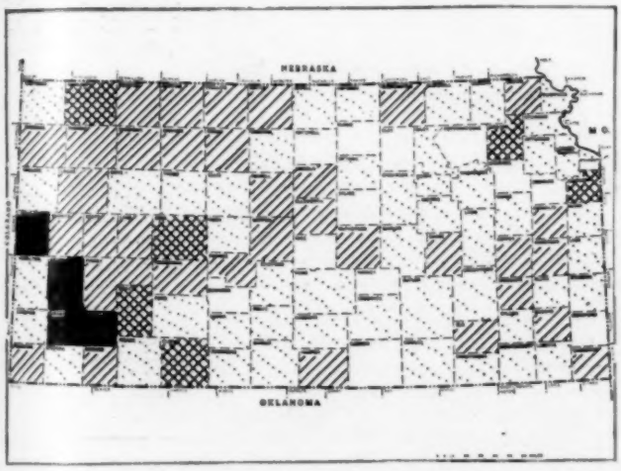
This is the second of a series of charts showing the trend in locations of physicians from April 1946 to January 1947; the first series appeared May 17, page 301.

"Upon the return of medical officers from the armed forces, the distribution of physicians in Kansas was greatly improved. Generally speaking, the larger communities in the state are adequately supplied with physicians at present. Requests are still being received from less populated areas, but these also are being filled.

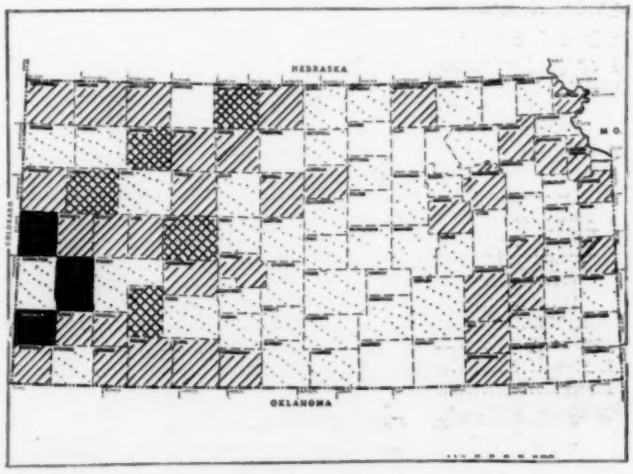
"At present there are thirty or forty communities in Kansas that have requested a resident physician. In almost all instances those communities are near larger cities where medical care is available. Frequently these communities have less than 200 population and could not in themselves adequately support a doctor. There are, however, some areas where the physician-patient ratio is still below normal. There is one community in Kansas with a population of more than 1,000 that does not have a resident physician. Many of the smaller Kansas towns are contemplating the construction of hospitals. If these projects are completed, it appears that those communities will become more attractive to doctors and at that time the needs that exist today will gradually be filled."

MR. O. E. EBEL, Executive Secretary.
Kansas Medical Society,
512 New England Building,
Topeka, Kan.

KANSAS—1946



KANSAS—1947



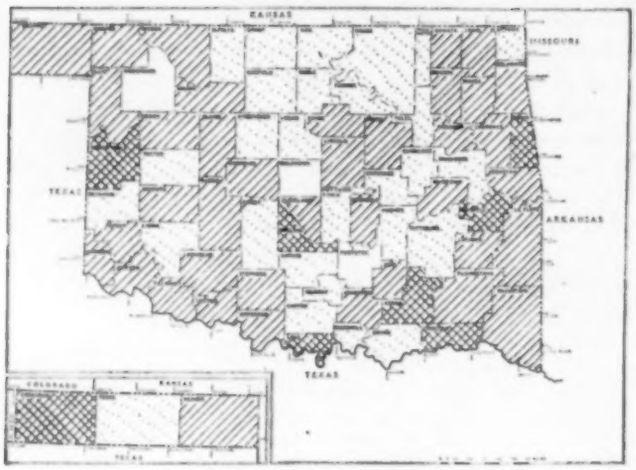
Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

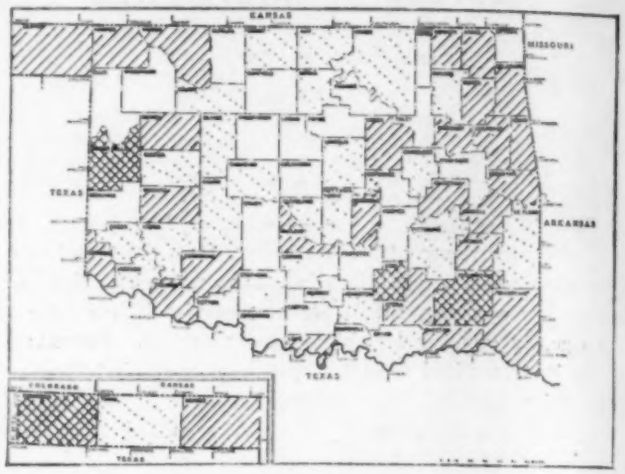
Areas in Kansas Requesting General Practitioners

County	Town	Population
Allen	Elsmore	180
Anderson	Greeley	387
	Kincaid	319
Atchison		22,222
Butler	Whitewater	515
Chautauqua	Elgin	336
Cherokee		29,817
	Scannon	737
Cloud	Glasco	741
	Jamestown	490
Dickinson	Enterprise	706
Elk		8,160
	Longton	479
Gove	Quinter	455
Graham		6,071
Grant		1,946
	Ulysses	824
Greenwood	Severy	393
Hamilton		2,645
Jefferson		12,718
	Nortonville	700
Kearny		2,525
Kingman	Norwich	411
Kiowa	Haviland	443
Labette	Edna	500
	Oswego	1,953
Lincoln	Vesper	150
Logan	Oakley	1,136
Marion	Burns	409
	Florence	1,329
Marshall	Irving	350
	Vermillion	309
Meade	Fowler	514
Morris	Council Grove	2,723
Nemaha	Centralia	607
	Goff	339
	Wetmore	425
Neosho	Thayer	469
Osage	Carbondale	415
	Overbrook	575
	Quenemo	564
Osborne		9,835
	Natoma	651
Phillips	Kirwin	395
Rawlins	Herndon	400
Reno	Pretty Prairie	404
Republic	Agenda	178
Rice	Little River	537
Riley	Riley	392
Stanton		1,443
	Johnson	524
Sumner	Belle Plaine	878
Wabaunsee		9,219
Wallace	Sharon Springs	760
Washington	Clifton	670
Wichita		2,185
	Leoti	816

OKLAHOMA—1946



OKLAHOMA—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

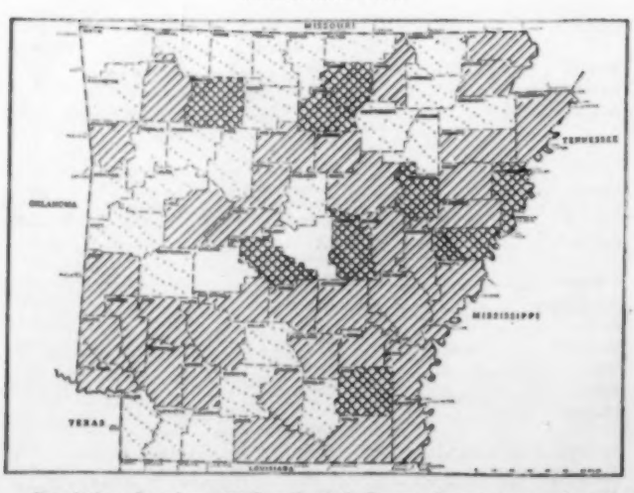
Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in Oklahoma Requesting General Practitioners

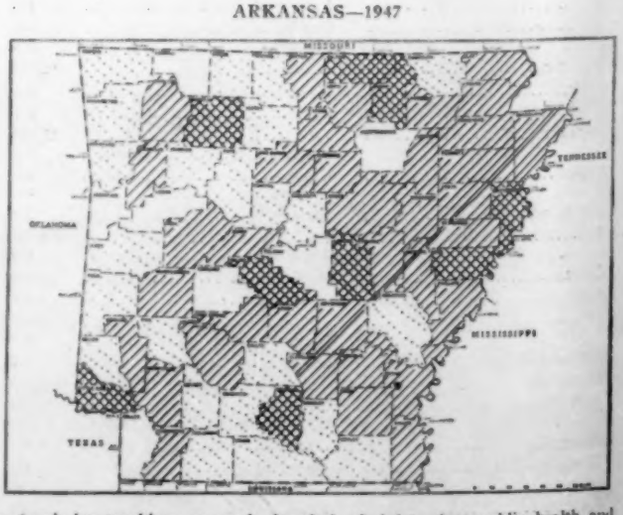
County	Town	Population	County	Town	Population
Alfalfa	Aline	405	Logan		25,245
Atoka	Atoka	2,548	McClain	Wayne	401
Blaine		18,543	Murray	Davis	1,698
Bryan	Durant	10,027		Sulphur	4,970
Choctaw	Hugo	5,900	Osage	Fairfax	2,327
Cleveland	Norman	11,429	Ottawa	Afton	1,261
Custer	Butler	429		Miami	8,345
Grady	Chickasha	14,111	Payne	Ripley	415
Jefferson	Ryan	1,115		Yale	1,407
Kiowa	Mountain View	1,075	Pontotoc	Ada	15,143
LeFlore		45,806	Washington		30,550
Lincoln		29,520	Woods	Waynoka	1,584
	Chandler	2,738	Woodward	Mooreland	811

Mr. Dick Graham, Secretary,
Oklahoma State Medical Association,
210 Plaza Court,
Oklahoma City 3.

ARKANSAS—1946



ARKANSAS—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

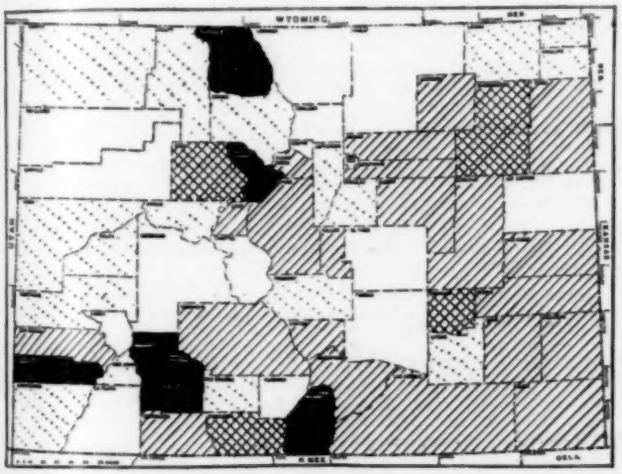
Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in Arkansas Requesting General Practitioners

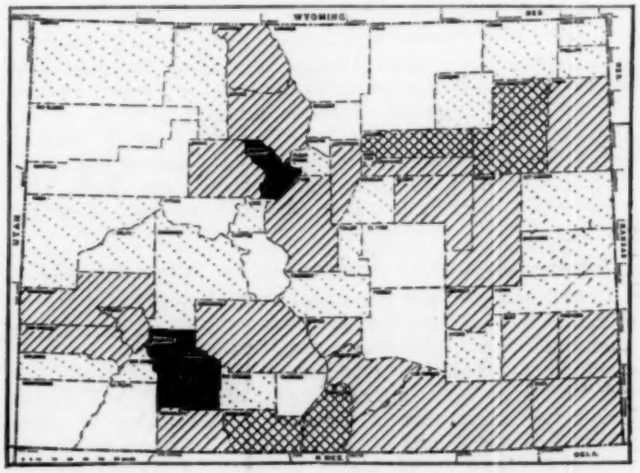
County	Town	Population
Arkansas		24,437
Ashley	Montrose	343
Benton		36,148
	Decatur	3,502
Clark		24,402
Cleveland		12,570
Craighead	Nettleton	909
Crawford		23,920
Grant		10,477
Hot Spring	Bismarck	75
Independence	Newark	802
Izard	Calico Rock	738
	Melborne	567
Lawrence		24,437
	Alicia	333
	Imboden	525
	Strawberry	110
Lee		26,810
Lincoln	Star City	1,090
Mississippi		80,217
Monroe		21,133
	Brinkley	3,409
Montgomery		8,876
Pope		25,682
	London	418
Phillips		45,970
St. Francis	Widener	225
Scott		13,200
Sevier	DeQueen	3,055
Sharp		11,497
	Evening Shade	347
Stone		8,693
	Mountain View	745
Van Buren		12,518
Washington	Elkins	243
	Lincoln	720
	Winslow	248
Woodruff	Cotton Plant	1,778
Yell		20,970
	Havana	449

DR. W. R. BROOKSHER, Secretary,
Arkansas Medical Society,
602 Garrison Avenue,
Fort Smith, Ark.

COLORADO—1946



COLORADO—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in Colorado Requesting General Practitioners

County	Town	Population
Adams	Brighton	4,029
Baca	Walsh	406
Dolores		1,958
Douglas		3,496
Eagle		5,361
	Minturn	596
Elbert		5,460
Garfield		10,560
Gilpin		1,625
Jackson		1,798
Lake		6,883
Lincoln		5,882
	Limon	1,053
Montezuma		10,463
Montrose	Montrose	4,764
Saguache	Saguache	1,219
San Miguel	Norwood	412
Summit	Breckenridge	381
Washington	Akron	1,417
Weld	Ault	761
	Keenesburg	284

"The distribution of physicians in Colorado is still too heavily concentrated in the large and medium size cities if the state is taken as a whole. So long as the current general prosperity lasts I believe that cities such as Denver, Colorado Springs, Pueblo, Greeley, Fort Collins, Boulder and Grand Junction can adequately support the large number of physicians now practicing in these locations, but if a recession or a genuine depression sets in, as many economists predict, I feel that some or all of the cities named will find themselves overstaffed with physicians. There still are fifteen or twenty excellent locations for general practitioners in the very small communities and rural areas of Colorado and probably another fifteen or twenty locations of this type which might be called good rather than excellent. Three or four such communities might be considered in critical need. Within the last six months many previously critical locations in Colorado have been filled, indicating somehow that a better appreciation of the worth of rural practice has developed among some physicians. Financial consideration seems to have little or no bearing on failure of physicians to fill the remaining critical locations in Colorado; rather, failure of physicians to accept these locations is due to lack of hospital facilities and lack of good general living conditions. With the increased interest of local communities in developing hospital facilities, with or without the aid of Public Law 725 (Hill-Burton Hospital Survey and Construction Act), I consider the Colorado situation hopeful for filling remaining critical areas within the next year."

MR. HARVEY T. SETHMAN, Executive Secretary,
Colorado State Medical Society,
537 Republic Building,
Denver 2.

Washington Letter

(From a Special Correspondent)

May 29, 1947.

General Bradley Opposes Giving Free Automobiles to Amputee Veterans

Gen. Omar Bradley, Veterans Administrator, has registered a protest with Chairman Edith Nourse Rogers of the House Veterans Committee opposing legislation to give free automobiles to veterans who lost their sight or suffered arm amputations during the war. He warned that, "by logical progression, such a move might ultimately demand that all seriously disabled veterans be supplied with automobiles, in addition to compensation and other benefits." He estimated the cost of giving cars to all amputees and blind veterans at $21,930,000. Congress has already appropriated $30,000,000 for a program under which leg amputees now get cars at government expense provided they cost no more than $1,600. War amputees demonstrated new artificial limbs for President Truman, Secretary of War Patterson and General Bradley.

International Council of Nurses Holds Precongress Meeting

Delegates to the ninth congress of the International Council of Nurses in Atlantic City, May 11 to 16, met first in Washington, where they attended a tea at the White House as guests of Mrs. Truman and conducted business sessions. More than 5,000 nurses from thirty-two countries, 500 of them from overseas, will be at the congress. Speakers declared that the nursing profession is "only at the threshold of its potential value to the good health of the world tomorrow." Delegates also observed the one hundred and twenty-seventh anniversary of the birth of Florence Nightingale (May 12, 1820), founder of modern nursing.

Interracial Medical Forum Held in National Capital

Described as the first interracial medical program of its kind ever held in Washington, a postgraduate medical assembly was conducted under sponsorship of the District of Columbia Physicians Forum to discuss "Problems of the Aging Patient." Dr. Ernst Boas, assistant clinical professor of medicine at Columbia University, was chairman. Speakers included Drs. W. Montague Cobb and Charles Drew of Howard University Medical School, Benjamin I. Weininger, president of the Washington-Baltimore Psychoanalytic Society, Charles Geschikter, professor of pathology at Georgetown University Medical School, and Frederick Zeman, director of the Home for Aged and Infirm Hebrews, New York.

Dr. John Minor Heads District Medical Society

Dr. John Minor, specialist in internal medicine, was elected president of the District of Columbia Medical Society, succeeding Dr. Raymond T. Holden. Other officers elected were William P. Herbst Jr. to the executive board, Iva A. Pelzman first vice president, Carolyn Pincok second vice president, W. Ross Morris and William W. Chase to the board of censors, Coursen B. Conklin delegate to the American Medical Association and Worth B. Daniels and J. Lawn Thompson Jr. alternate delegates to the American Medical Association.

Navy Consents to Use of Observatory Land for Medical Center

The Navy Department revealed that it is willing to move the Naval Observatory, between Wisconsin and Massachusetts avenues, to a new site to make way for the proposed new $20,000,000, 1,500 bed medical center planned for the nation's capital. The new center would incorporate three local hospitals, Garfield, Emergency and Episcopal. The last Congress authorized an appropriation of $35,000,000 for the overall project.

Modern Medicine in the Treatment of Alcoholism

Dr. Howard W. Haggard, Yale physiologist, informed the Washington Committee for Education on Alcoholism that modern medical methods are already successful in the treatment of alcoholic addicts. He said that 80 per cent of those who continue through the full period of treatment at the Yale clinic recover. Dr. Haggard spoke on "Alcoholism as a Medical Problem."

Coming Medical Meetings

American Medical Association, Atlantic City, June 9-13. Dr. George F. Lull, 535 N. Dearborn St., Chicago 10, Secretary.

Aero Medical Association of the United States, Atlantic City, Ritz-Carlton Hotel, June 4-6. Dr. David S. Brachman, 1016 Dime Bldg., Detroit 26, Secretary.

American Association for the Surgery of Trauma, Atlantic City, Hotel Claridge, June 5-7. Dr. Gordon M. Morrison, 520 Commonwealth Ave., Boston 15, Secretary.

American Association of Genito-Urinary Surgeons, Absecon, N. J., June 4-6. Dr. Charles C. Higgins, 2020 E. 93d St., Cleveland 6, Secretary

American College of Allergists, Atlantic City, Hotel Senator, June 6-8. Dr. Fred W. Wittich, 401 LaSalle Medical Bldg., Minneapolis 2, Secretary.

American College of Chest Physicians, Atlantic City, Ambassador Hotel, June 5-8. Mr. Murray Kornfeld, 500 N. Dearborn St., Chicago 10, Executive Secretary.

American College of Radiology, Atlantic City, Haddon Hall, June 7-8. Mr. Mac F. Cahal, 20 N. Wacker Drive, Chicago 6, Executive Secretary.

American Dermatological Association, Murray Bay, Quebec, Can., June 1-5. Dr. Harry R. Foerster, 208 E. Wisconsin Ave., Milwaukee, Wis., Secretary.

American Diabetes Association, Atlantic City, Chalfonte-Haddon Hall, June 7-8. Dr. Cecil Striker, 1019 Provident Bank Bldg., Cincinnati 2, Secretary.

American Geriatrics Society, Atlantic City, Hotel Brighton, June 5-7. Dr. Malford W. Thewlis, 25 Mechanic St., Wakefield, R. I., Secretary.

American Heart Association, Atlantic City, Hotel President, June 6-7. Dr. Howard B. Sprague, 50 W. 50th St., New York, Secretary.

American Medical Women's Association, Atlantic City, Hotel Claridge, June 7-8. Dr. Helen F. Schrack, 216 N. Fifth St., Camden, N. J., Secretary.

American Neisserian Medical Society, Atlantic City, Marlborough-Blenheim Hotel, June 8. Dr. William L. Fleming, 750 Harrison Ave., Boston, Secretary.

American Neurological Association, Atlantic City, Marlborough-Blenheim Hotel, June 16-18. Dr. Walter O. Klingman, 195 Ft. Washington Ave., New York 32, Secretary.

American Ophthalmological Society, Hot Springs, Va., June 5-7. Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y., Secretary.

American Physiotherapy Association, Pacific Grove, Calif., July 6-12. Miss Mildred Elson, 1790 Broadway, New York 19, Secretary.

American Proctologic Society, Atlantic City, June 7-8. Dr. Harry E. Bacon, 2031 Locust St., Philadelphia 3, Secretary.

American Radium Society, Atlantic City, June 8. Dr. Hugh F. Hare, 605 Commonwealth Ave., Boston 15, Secretary.

American Rheumatism Association, Atlantic City, Ritz-Carlton Hotel, June 7. Dr. Richard H. Freyberg, 321 E. 42d St., New York 17, Secretary.

American Society for the Study of Sterility, Atlantic City, Hotel Strand, June 7-8. Dr. John O. Haman, 490 Post St., San Francisco 2, Secretary.

American Society of Electroencephalography, Atlantic City, Marlborough-Blenheim Hotel, June 13-14. Dr. Robert S. Schwab, Massachusetts General Hospital, Boston 14, Secretary.

American Therapeutic Society, Atlantic City, Chalfonte Hotel, June 6-7. Dr. Oscar B. Hunter, 1835 I St., N. W., Washington 6, D. C., Secretary.

American Urological Association, Buffalo, N. Y., Hotel Statler, June 30-July 3. Dr. Thomas D. Moore, 899 Madison Ave., Memphis 3, Tenn., Secretary.

Association for the Study of Internal Secretions, Atlantic City, June 6-7. Dr. Henry H. Turner, 1200 N. Walker St., Oklahoma City 3, Secretary.

Idaho State Medical Association, Sun Valley, June 16-19. Dr. William B. Handford, Caldwell, Secretary.

Maine Medical Association, York Harbor, Marshall House, June 22-24. Dr. Frederick R. Carter, 142 High St., Portland 3, Secretary.

Minnesota State Medical Association, Duluth, June 30-July 2. Dr. B. B. Souster, Lowry Medical Arts Bldg., St. Paul 2, Secretary.

Montana, Medical Association of, Missoula, June 26-28. Dr. H. T. Caraway, 115 N. 28th St., Billings, Secretary.

National Gastroenterological Association, Atlantic City, Hotel Chelsea, June 4-6. Dr. G. Randolph Manning, Room 319, 1819 Broadway, New York, Secretary.

National Tuberculosis Association, San Francisco, June 16-19. Dr. F. D. Hopkins, 1790 Broadway, New York 19, Executive Secretary.

New Hampshire Medical Society, Portsmouth, Hotel Wentworth, June 19-20. Dr. Carleton R. Metcalf, 5 South State Street, Concord, Secretary.

South Dakota State Medical Association, Rapid City, May 31-June 3. Dr. Roland G. Mayer, 22½ S. Main St., Aberdeen, Secretary.

Wyoming State Medical Society, Sheridan, June 23-25. Dr. George E. Baker, 226 E. 2d St., Casper, Secretary.

Medical Legislation

STATE LEGISLATION

Alabama

Bills Introduced.—H. 136 proposes to authorize counties to **levy a tax** for the purpose of acquiring, constructing, operating, equipping or maintaining county or other public hospitals and public health facilities. H. 141 and S. 90 propose the enactment of a **hospital planning and construction act** to provide funds to the state board of health for the planning and construction of state owned and other public and nonprofit hospitals in the state. H. 151 and S. 97, to amend the **law relating to marriages**, propose that each applicant for a marriage license shall file a certificate signed by a legally licensed physician in Alabama, or in the state where the applicant resides, certifying that the applicant is not infected with syphilis in a communicable stage. The present law requires such an examination for males only. S. 36 proposes an additional appropriation to the **University of Alabama Medical College** in the sum of $56,750 each for two years. S. 102 proposes the establishment of **an advisory council** to consult with the state board of health in planning for, acquiring, constructing, equipping, maintaining and supervising hospitals, health centers and related facilities for the treatment of disease.

California

Bill Introduced.—S. 763 proposes the enactment of a law regulating **health care** expense contractors.

Bills Passed.—A. C. A. 23, which passed the house May 13, proposes an **amendment to the constitution** to the effect that no provision in the constitution shall be construed as a limitation on the power of the legislature by general law to give or lend state money or other assistance for the construction of hospitals, health centers and related facilities to counties, cities and counties, cities, local hospital districts, other public districts authorized to construct hospitals, etc. A. 2428, which passed the house May 14, to amend the business and professions code, proposes under certain circumstances to authorize licentiates of other states to act as **resident or assistant resident physicians** and surgeons in state hospitals for a period of not exceeding one year.

Bill Enacted.—S. 250, which has become chapter 234 of the Laws of 1947, repeals the existing law relating to the examination and licensure of **trained hospital attendants.**

Connecticut

Bill Passed.—S. 170, which passed the house May 19, proposes to broaden the scope of activities of the **bacteriologic laboratory** so as to conform with those of a modern public health laboratory.

Florida

Bills Introduced.—H. 257 has previously been reported as proposing a law for the licensing, inspecting and regulating of hospitals and the creation of **an advisory hospital council.** Two amendments were added to this bill by the house committee on May 13. The first would exempt from its provisions the establishment or operation of any hospital for the practice of the religious tenets of any church in the ministration to the sick and suffering by mental or spiritual means without the use of any drug or remedy, provided the statutes or regulations on sanitation are complied with. The second would prohibit the rule making authority from making any rule which would have the effect of denying a license to a hospital or other institution solely by reason of the school or system of practice employed or permitted to be employed by physicians therein, provided that such school or system of practice is recognized by the laws of the state. H. 815 proposes to authorize the **appointment of medical examiners** and assistant medical examiners in each county to perform functions similar to those of a coroner. S. 614 proposes to require the state board of health to develop and maintain **a state blood program** for the purpose of coordinating and integrating the services of autonomous, nonprofit blood banks, to organize additional blood banks and to establish subsidiary blood banks and distribution centers in order that these therapeutic products shall be made available at hospitals and to all physicians licensed to practice in Florida and to insure that the supply of blood and blood products shall be adequate to meet reasonable requirements.

Bills Passed.—H. 253, which passed the senate May 14, proposes to authorize persons to donate and give their eyes for **eyesight restoration** purposes by last will and testament and authorizes any state, county, district or other public hospital to purchase and provide the necessary facilities and equipment to establish and maintain an eye bank for restoration of sight purposes. H. 440, which passed the house May 15, proposes to make it unlawful for tax collectors in the several counties to issue **state and county occupational licenses** to any person applying for license to practice medicine in any of its branches unless and until proof of current qualification and competency, as established by certificate or license issued by the state boards duly constituted and legally authorized to determine qualification and competency, be exhibited at the time of making such application. S. 151, which passed the house May 9, to amend the law relating to the **practice of massage**, proposes to redefine the meaning of the practice so as to include the use of electrical apparatus.

Maine

Bills Enacted.—S. 164, which has become chapter 191 of the Laws of 1947, creates a commission to investigate and study the feasibility and desirability of establishing **a medical school in the state of Maine.** S. 449, which has become chapter 391 of the Laws of 1947, authorizes the governor to appoint **an advisory hospital council** to advise and consult with the department of health and welfare in carrying out the administration of the existing hospital survey act.

Michigan

Bill Passed.—H. 451, which passed the house May 16, proposes the enactment of a law requiring the licensing, regulation and **inspection of hospitals** and related institutions and the appointment of an advisory institutional council.

Missouri

Bill Passed.—S. 157, which passed the senate May 14, to amend the sales tax act, proposes that purchases of **tangible personal property** made by duly licensed physicians, dentists and veterinarians and used in the practice of their professions shall be deemed to be purchases for use or consumption and not for resale.

New Hampshire

Bill Introduced.—H. 456 proposes the creation of **a mental hygiene commission** to manage and conduct mental hygiene clinics primarily for the psychiatric and psychologic study and treatment of such children as may be committed to its care.

Oklahoma

Bills Enacted.—S. 76, which was approved May 16, requires every prospective **teacher in the public schools** to have a medical examination for the purpose of determining physical qualifications in accordance with regulations prescribed by the state board of health. S. 100, which was approved May 16, creates within the state health department a division to be known as the **Division of Preventive Dentistry** to administer a program of dental care, consultation, research and health education designed to improve the dental health of the people of the state.

Pennsylvania

Bills Passed.—H. 1043, which passed the house May 13, proposes, among other things, to prohibit the **sale of benzedrine,** and other, inhalers at retail. S. 335, which passed the house May 14, to amend the law relating to **consumer credit,** proposes that the act shall not apply to nonprofit corporations organized under the laws of the state having memberships consisting exclusively of duly licensed physicians, surgeons and dentists operated exclusively for and by their members and engaged in, among other activities, the financing of personal obligations arising from the rendering of professional services by the members of such nonprofit corporations which are limited strictly to medical, surgical, dental, operative, diagnostic and treatment services or nursing and hospital care.

South Carolina

Bills Enacted.—S. 254, which has become chapter 399 of the Laws of 1947, provides that **osteopathic physicians** duly licensed to practice their profession under the laws of the state are authorized and shall certify birth and death certificates in the same manner as is now prescribed by law for other physicians. S. 721, which has become chapter 511 of the Laws of 1947, creates a combination state **hospital construction act** and hospital licensing law.

Texas

Bill Introduced.—H. 833 proposes to authorize the **state board of health** to receive gifts, donations and contributions from various sources for the purpose of building, constructing, maintaining and operating a medical research laboratory.

Bills Passed.—H. 138, which passed the house May 5, proposes the creation of a state board of **examiners in the basic sciences.** A house floor amendment of May 1 proposes to exempt from the basic science law any chiropractor who is a graduate of a reputable chiropractic college in the United States and who has practiced chiropractic for three years immediately preceding the effective date of this act and who has resided in Texas for two years immediately preceding the effective date of this act and who has never had a license to practice any branch of the healing art canceled by any American or Canadian state, province or territory. H. 503, which passed the house May 6, proposes the enactment of a state hospital survey and construction act requiring **a statewide survey of hospital facilities** and the preparation of a program for the construction of additional needed facilities. The proposal also requires the appointment of an advisory hospital council and the establishment of regulations for the purpose of obtaining federal funds to assist in survey and construction programs. The administration of this proposal would be under a division of hospital survey and construction in the department of health. S. 120, which passed the senate April 28, to amend the penal code, proposes to make it unlawful for any physician or other practitioner of the art of healing **to advertise special skills,** definite prices or the use of superior qualities of medicine, material, devices, etc. in the performance of professional service.

Wisconsin

Bills Passed.—S. 104, which passed the senate May 14, proposes regulations for the licensing, inspecting and **regulating of hospitals** within the state. S. 105, which passed the house May 19, proposes the enactment of a **state hospital survey and construction act** requiring a survey of hospitals and health facilities in the state and the preparation of a program for the construction of additional needed facilities. The proposal would require the appointment of an advisory hospital council and would authorize the state board of health to apply for and receive federal funds to assist in such survey or construction program. S. 156, which passed the house May 19, to amend the law relating to the **licensing of osteopaths,** proposes to require applicants for a license to practice osteopathy to have completed at least twelve months' internship in a reputable osteopathic hospital or its equivalent.

GOVERNMENT SERVICES

PUBLIC HEALTH SERVICE

NEW DIVISION OF TROPICAL DISEASES

The Surgeon General, Dr. Thomas Parran, announces that the Public Health Service has established a new division of tropical diseases in the National Institute of Health, Bethesda, Md. Heading the new unit will be Willard H. Wright, formerly chief of the zoology laboratory and, during the war, field director of the Commission on Schistosomiasis in the Southwest Pacific and Japan. Dr. Wright received his D.V.M. degree from George Washington University in 1917 and a Ph.D. in 1935. He is a member of the American Academy of Tropical Medicine, the American Society of Parasitologists, the Helminthological Society of Washington and the New York Academy of Sciences.

NEW CHIEF OF THE OFFICE OF DERMATOLOGY

Paul C. Campbell Jr., Surgeon, has been appointed chief of the Office of Dermatology, Industrial Hygiene Division, U. S. Public Health Service, succeeding Dr. Louis Schwartz, who will retire July 4 after forty-one years of service. Dr. Campbell, who has been on duty in the office of dermatology since 1944, has been investigating dermatitis hazards in various industries. Before entering the Public Health Service in 1941 he was for several years a resident in dermatology and syphilology at the Edward J. Meyer Memorial Hospital in Buffalo.

PERSONAL

Medical Director (R) Russell S. Ferguson, Washington, D. C., has been ordered to active duty and to report to the U. S. Marine Hospital, San Francisco.

CALLED TO ACTIVE DUTY

The U. S. Public Health Service reports that the following officers have been called to active duty in either the reserve corps or the regular corps and have been directed to report to the stations opposite their names:

Azulay, Abraham, assistant surgeon (R), U. S. P. H. S., Medical Center, Hot Springs National Park, Ark.

Cohart, Edward M., surgeon (R), U. S. P. H. S., District 1, 15 Pine St., New York.

Davenport, Dennis D., assistant surgeon (R), District Coast Guard Office, Seattle.

Green, James R., assistant surgeon (R), U. S. Marine Hospital, Memphis, Tenn.

Mahe, George A., Jr., assistant surgeon (R), U. S. Penitentiary, Atlanta, Ga.

Peck, James F., assistant surgeon (R), U. S. P. H. S., Medical Relief Station, Louisville, Ky.

Velat, Clarence A., assistant surgeon (R), U. S. P. H. S., States Relations Division, Washington, D. C., Bethesda Station.

OFFICERS RELEASED FROM SERVICE

U. S. Public Health Service headquarters reports that the following medical officers have been terminated, inactivated or retired and directed to proceed to the city opposite their name:

Abrams, Henry, surgeon (R), Princeton, N. J.
Ausubel, David P., assistant surgeon (R), Bronx, N. Y.
Kintner, Burton E., S. A. surgeon (R), Elkart, Ind.
Rodman, Peter P., S. A. surgeon (R), Aberdeen, Md.
Rothfeld, Edmond, S. A. surgeon (R), Cincinnati.
Warhaftig, Melvin V., assistant surgeon (R), Hillside, N. J.

MISCELLANEOUS

DISTRIBUTION OF "HEAVY WATER"

The United States Atomic Energy Commission has announced plans for the distribution of heavy water and deuterium gas to qualified research institutions in the United States.

Heavy water was produced in connection with the atomic energy operations and now is available in sufficient quantities for domestic research purposes outside the government's atomic energy program. The Stuart Oxygen Company of San Francisco, which will make the distribution under contract with the commission, will convert heavy water to deuterium gas for applicants who need deuterium in the latter form.

Applicants for deuterium oxide (heavy water) and deuterium gas will be charged an amount sufficient to cover handling and distribution charges of the contract distributor. The price schedule for heavy water is 50 cents per gram for the first hundred grams and 30 cents per gram thereafter and for deuterium gas $1 per liter (at normal temperature and pressure) for the first hundred liters and 80 cents per liter thereafter. Prices are based on orders calling for single deliveries to the applicant.

Applications will be handled in the same manner as requests for radioactive isotopes produced in the pile at Oak Ridge and distributed by the Monsanto Chemical Company. Application forms can be obtained from the Atomic Energy Commission, Research Division, Isotopes Branch, Oak Ridge, Tenn. Applications will be screened by the subcommittees of the Advisory Committee on Isotope Distribution in order to insure the most efficient use of the materials. Approved applications will be returned to the applicant for transmittal to the contract distributor. Users, as in the case of radioisotopes, will be required to furnish the commission reports on the results of experiments carried on with heavy water and deuterium. Applicants must agree not to transfer the materials to another user without the consent of the commission.

Heavy water contains the heavy stable isotope of hydrogen referred to as deuterium and is symbolized by D_2O. It is found in ordinary water at a ratio of 1 to every 4,500 parts of ordinary water. The heavy hydrogen isotope has twice the atomic mass of the ordinary hydrogen atom.

Heavy water and deuterium have numerous uses in physics, chemistry and biology. In nuclear physics deuterium is of special value and has a number of uses:

1. High voltage accelerators such as the cyclotron use deuterium as a source of bombarding particles in studying nuclear reactions. Deuterium bombardment of light elements results in a large yield of high energy neutrons.

2. Heavy water is an effective moderator, serving to slow down the fast neutrons. Ordinary water will also slow down fast neutrons but absorbs a large proportion of the neutrons. The relatively small neutron absorption of heavy water makes it useful as a moderator in certain types of chain reacting piles. Amounts made available under this plan of distribution will permit research on the proportion of heavy water as a moderator.

3. Heavy water is used in determining the energy of gamma rays through photo production of neutrons. This is useful in studying the energy of gamma rays.

Deuterium is extensively used as a tracer for hydrogen in biologic and chemical processes. Hydrogen has been used to study the metabolism of normal and malignant cells in attempts to discover the nature and causes of cancer. Heavy water can also be used as a tracer in studying the exchange of water in various systems; for example, between a fish and its environment. It can also be used to study the fluid balance in man.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Fund to Fight Heart Disease.—A committee representing the Los Angeles County Tuberculosis and Health Association will jointly administer the A. M. Roberts Memorial Fund to fight heart disease. The fund was established through the efforts of the late Dr. Archie M. Roberts, formerly of the University of Southern California School of Medicine, Los Angeles, and until his death, April 1, president of the Los Angeles Heart Association. Contributions will be used for the care of children with rheumatic fever and other heart ailments and for research.

Changes in Health Officers.—Recent additions to the staff of the state department of public health include Dr. James W. Ellis, Washington, D. C., who will be cancer consultant with the bureau of chronic diseases. Dr. Ellis comes to the department following thirty years of service with the navy. He is a graduate of the University of Pennsylvania School of Medicine, Philadelphia, 1915, was certified by the American Board of Surgeons and was medical director in the U. S. Navy with the rank of captain. Dr. Harvey F. Hendrickson, San Francisco, has been appointed to the bureau of hospital inspection as an assistant to Dr. Julius B. Askew. Dr. Hendrickson, a graduate of the University of Pennsylvania School of Medicine, Philadelphia, 1927, was a major in the medical corps of the army. During the past year he has been assistant superintendent of the San Francisco Hospital.

COLORADO

Rocky Mountain Cancer Conference.—This conference will be held in Denver July 9 and 10 at the Shirley-Savoy Hotel with the following program:

Dr. Ashley W. Oughterson, New York, Aims of the American Cancer Society.
Dr. Hugh J. Morgan, Nashville, Tenn., Importance of History and Physical Examination in the Recognition of Cancer.
Dr. Harry C. Solomon, Boston, Psychiatric Implications of Cancer.
Dr. Shields Warren, Newton, The Radiosensitivity of Tumors.
Dr. Edwin C. Ernst, St. Louis, Use of the Roentgen Ray in the Treatment and Diagnosis of Cancer.
Dr. Frank Adair, New York, Diagnosis and Treatment of Breast Tumors.
Dr. Charles B. Huggins, Chicago, Carcinoma of the Prostate.
Dr. Eugene F. Traub, Jackson Heights, N. Y., The So-Called Premalignant Eruptions of the Skin.
Dr. Joe Vincent Meigs, Brookline, Mass., Carcinoma of the Lip and Mouth.

CONNECTICUT

Rural Health Conference.—The Connecticut State Medical Society and the University of Connecticut will sponsor a rural health conference at the university on July 16. Members of the society's rural health committee now preparing plans for the conference include Drs. Norman H. Gardner, East Hampton, chairman; Gert S. Gundernatsch, Sharon; David H. Bates, Putnam, and William H. Upson, Suffield.

Dr. Walker Appointed to Public Health Council.—The governor recently appointed Dr. W. Bradford Walker, Cornwall, to the Connecticut Public Health Council, effective July 1, for a term of six years. Formerly president of the Connecticut Public Health Association and past president of the Litchfield County Medical Association, Dr. Walker will succeed Dr. David R. Lyman, who is retiring from the council following eighteen years of continuous membership. Dr. Lyman is a past president of the Connecticut State Medical Society, the New Haven County Medical Association, the American Sanatorium Association and the National Tuberculosis Association.

ILLINOIS

The Fifty-Year Club.—Fifty years of service by each of five Illinois physicians have been recognized by Illinois county medical societies. Champaign County Medical Society presented certificates of membership in the "Fifty-Year Club" and gold buttons signifying fifty years of medical service to Drs. William E. Schowengerdt, Champaign; Charles H. Spears, Champaign; Emerson M. Brewer, Rantoul, and Joseph Brayshaw, Homer. At Mount Vernon Dr. Moss Maxey, at a dinner given by the Jefferson County Medical Society May 8, was likewise honored. Membership in the club is awarded by the Illinois State Medical Society through county organizations.

New General Hospitals.—Formal opening and public inspection of the new $150,000 Milan General Hospital, Milan, took place April 13 with religious, political and civic leaders in attendance. The hospital has been under construction for more than a year. Dr. Edwin F. Condon is president and director. ——A $1,400,000 building program will begin in August for the expansion of MacNeal Memorial Hospital, Berwyn. The present capacity will be doubled, and quarters for nurses will be provided. The new addition will be four stories in height, except a center portion which will be five stories. A new suite of operating rooms, an enlarged x-ray department, delivery and labor rooms and other laboratories will be added.

Chicago

Hilton Hotels Appointment.—Richard L. Collison, director of sales of the Palmer House, has been appointed director of sales for the entire Hilton Hotel chain. He will continue, however, to make his headquarters in Chicago.

Million Poliomyelitis Leaflets to be Sent to Parents.—The Cook County chapter of the National Foundation for Infantile Paralysis will distribute a million leaflets explaining precautions to be taken during the summer. The leaflets will be delivered to public and parochial schools throughout Chicago and Cook County for children to take home to their parents. Bulk distribution will also be made to large employers. A card included with the pamphlet urges that, if poliomyelitis occurs, a physician be called at once and that the Cook County chapter, National Foundation for Infantile Paralysis, be called if help is needed.

Symposium in Honor of Illinois President.—An inaugural symposium in honor of President George D. Stoddard, University of Illinois, was held May 15 in the lecture hall of the University of Illinois College of Medicine. Speakers were Dr. Roscoe R. Spencer, director of the National Cancer Research Institute, Bethesda, Md.; Dr. Edmund V. Cowdry, professor of anatomy and head of the department, Washington University School of Medicine, St. Louis, "Geriatrics," and Dr. Austin M. Brues, associate professor of medicine, University of Chicago Institute of Radiology and Biophysics, "Medical Implications of Atomic Research." Dr. Anton J. Carlson, professor emeritus of the University of Chicago, was a guest speaker at a luncheon.

KENTUCKY

Three Clinical Professors to Retire.—At the end of the school year three Louisville physicians will retire as clinical professors from the University of Louisville School of Medicine, Louisville: Dr. Irvin Abell, clinical professor of surgery, president of the American College of Surgeons and formerly President of the American Medical Association; Dr. Bernard Asman, clinical professor and head of the department of proctology, and Dr. Charles W. Hibbitt, clinical professor of obstetrics and gynecology. All three graduated in the class of 1897, and all have offices in the same building, where they will continue their private practice. As professors emeritus they will be consultants to the medical school faculty.

LOUISIANA

Ochsner Foundation Hospital.—Dedication exercises were held April 13 for the foundation hospital, recently purchased by the Alton Ochsner Medical Foundation, New Orleans. The former army hospital wards have been converted into individual cubicles for private patients. Seven patient buildings with 161 beds are now in operation. Eventual plans are for a capacity of 300 beds.

Poliomyelitis Preparedness Conference.—The Louisiana State Department of Health, the National Foundation for Infantile Paralysis and the Orleans chapter of the National Foundation jointly will hold a poliomyelitis preparedness conference at the St. Charles Hotel, May 30-31. Among the speakers at the two day session will be Dr. Waldo L. Treuting, New Orleans, Louisiana State Board of Health, "Public Health Regulations and Facilities for Treatment of Infantile Paralysis Cases"; Dr. Oliver P. Daly, director, Charity Hospital of Louisiana, "The Charity Hospital System in Louisiana"; Dr. Kenneth S. Landauer, Geneva, N. Y., director of medical care, National Foundation, "Diagnosis and Treatment of Acute Poliomyelitis." Saturday a symposium on infantile paralysis will be held, subjects being pediatric aspects, modes of spread, orthopedic considerations, nursing advisory service and end results. The American Red Cross will present nursing problems involved in an infantile paralysis epidemic.

MASSACHUSETTS

Retired Navy Officers Appointed Health Officers.—Dr. Robert E. S. Kelley, captain, U. S. Navy (retired), has been appointed to the Massachusetts Department of Public Health as physician in charge of the second mobile unit of the State Blood Donor Program. Dr. Walter J. Pennell, captain, U. S. Navy (retired), has been appointed health officer of the Northeastern District of the Massachusetts Department of Public Health. Dr. Paul Richmond, captain (MC), U. S. Navy (retired), Washington, D. C., has been appointed health officer of the Worcester district of the Massachusetts Department of Public Health.

Dr. Faulkner Appointed Dean at Boston University.—Dr. James M. Faulkner, Boston, will assume his duties as dean of the Boston University School of Medicine, June 1, replacing Dean Donald G. Anderson, Boston, newly appointed secretary of the Council on Medical Education and Hospitals of the American Medical Association. Dr. Faulkner received his medical degree at Harvard Medical School, Boston, in 1924. He was assistant resident physician at the Rockefeller Institute Hospital, New York, in 1926, and the Johns Hopkins Hospital 1926-1927. He was a consultant in medicine at the U. S. Marine Hospital in Boston and director of the first and third medical services at Boston City Hospital, as well as a professor of medicine at Tufts College Medical School, Boston.

NEVADA

State Board Resumes Reciprocity.—At the May 5 meeting of the Nevada State Board of Medical Examiners the board unanimously voted to rescind the rule of suspension of reciprocity and to resume reciprocity. It also voted to meet only twice a year, i. e., in May and November, at which time it will hold both written examinations and oral reciprocity examinations.

NEW JERSEY

Society Organized.—The New Jersey Obstetrical and Gynecological Society was organized by about 125 members of the Medical Society of New Jersey at its annual meeting in Atlantic City April 23. The organization elected the following officers: Drs. Samuel A. Cosgrove, Jersey City, president; Herschel S. Murphy, Roselle, president-elect; George B. German, Camden, vice president; Benjamin Daversa, Spring Lake, secretary, and Osborne F. Christensen, Hackensack, treasurer. The society expects to hold its second meeting in Jersey City in October.

Dr. Donohoe Honored.—The medical staff of Bayonne Hospital and Dispensary will present Dr. Lucius F. Donohoe, medical director, with a portrait in oil in recognition of his services to the hospital, at a testimonial dinner June 5. This year marks his twentieth anniversary as medical director and his thirtieth anniversary as president of the medical staff. Dr. Donohoe served with the A. E. F. in France in World War I as a lieutenant colonel in the Medical Corps. He was commended in a citation for meritorious service by the American Expeditionary Forces. The republic of France conferred on him the Citation and Certificate of the Order of University Palms, grade of Officer of Academy, and later made him a Chevalier of the Legion of Honor.

NEW YORK

Sesquicentennial of Westchester County Society.—The Medical Society of the County of Westchester marked the one hundred and fiftieth consecutive year of its existence May 8. The society's sesquicentennial year will be commemorated by the publication of a volume of the history of the society to date. Dr. Laurence D. Redway, Ossining, will prepare the volume, which will be distributed at the annual meeting in November.

Professor Wiggers Comes to Albany Medical College.—Harold C. Wiggers, Ph.D., Chicago, will take over duties as head of the department of physiology and pharmacology at Albany Medical College July 1. The 38 year old teacher and research worker has been associate professor of physiology at the University of Illinois College of Medicine, Chicago, since 1943. He has done research on the coronary circulation, cardiodynamics, ovulation potentials and bioelectric phenomena. Dr. Wiggers received his bachelor's degree from Wesleyan University in 1932 and his doctor of philosophy from Western Reserve University in 1936. At Harvard Medical School, Boston, he held the W. T. Porter Fellowship. He was an instructor at Columbia University College of Physicians and Surgeons, New York, from 1937 to 1942 and was assistant professor at Western Reserve School of Medicine in 1942-1943.

Dr. Godfrey Retires.—Dr. Edward S. Godfrey Jr., state commissioner of health, retired May 1 after nearly thirty years in the state department of health. After graduation from the University of Virginia Department of Medicine, Charlottesville, he served both Arizona and Illinois before becoming epidemiologist in the New York State Department of Health. In 1920 he was appointed director of the division of communicable diseases and in 1931 director of local health administration. From 1928 to 1935 he served part time as clinical professor of epidemiology at the Columbia University College of Physicians and Surgeons. In 1936 he was appointed state commissioner of health. Dr. Godfrey has contributed many articles to the literature on public health, notably the report of a study of measles in children's institutions in the state outside of New York City. He is a member of numerous public health and medical societies.

New York City

Proposed Study of Rheumatic Heart Disease.—New York University College of Medicine in cooperation with the City Health Department will make a study of rheumatic fever and rheumatic heart disease among school children in the Lower East Side district next fall. The objective will be to develop efficient detection, follow-up and supervision which may be adopted on a citywide basis by the department of health. The study will be directed by Dr. Elvira M. DeLiee. Financial support comes from the New York Heart Association, U. S. Public Health Service, New York Department of Education, Rheumatic Fever Council of the American Heart Association, U. S. Children's Bureau, New York School of Social Work, Metropolitan Life Insurance Company and other departments of the university.

Master Plan for City Hospitals.—The hospital council of greater New York announced its master plan for city hospitals and related facilities last month. Based on the city's estimated 8,000,000 population requirements of 1950, the plan calls for 129,000 hospital beds, 33,600 for general care, 8,000 for convalescent care, 16,000 for long term illnesses, 800 for acute communicable diseases, 6,600 for tuberculosis and 64,000 for psychiatric care. It calls for eight regional hospitals and sixty-four community hospitals. The plan was drawn with the idea that distribution and location of hospital facilities should be determined by the needs of the people, the need for teaching and training programs and the need for medical research. The study has been endorsed by Mr. Edwin A. Salmon, chairman of the City Planning Commission, and Drs. John B. Pastore, executive director of the Hospital Council; George Baehr, president of the New York Academy of Medicine; William B. Rawls, representing the medical societies of the city; Claude W. Munger, director of St. Luke's Hospital; Edward M. Bernecker, city commissioner of hospitals, and Mr. Arthur A. Ballantine, chairman of the United Hospital Fund and the Greater New York Fund.

PENNSYLVANIA

Second Councilor District Meeting.—A meeting of the second councilor district of the Medical Society of the State of Pennsylvania will be held June 4 in the Masonic Temple, Allentown, at 12 noon. On the scientific program Dr. Chauncey L. Palmer, Pittsburgh, will speak on "Legislative Trends in the 1947 Session of the Pennsylvania Legislature"; Dr. Howard K. Petry, Harrisburg, president of the state society, "Indications for and Technic of Electro Shock," and David R. Perry Harrisburg, secretary to the governor, "Medicine Today and Tomorrow." Dr. Petry will present fifty year testimonials to the honor guests: Drs. Andrew Godfrey, Montgomery County; Chester K. Kistler, Berks County; William C. LeCompte, Bucks County; John B. Roxby, Delaware County, and Charles H. Schlesman, Lehigh County.

Seventh District Meeting.—The Seventh Councilor District Meeting of the Medical Society of the State of Pennsylvania was held May 9 at Williamsport with about 135 doctors and members of Woman's Auxiliaries in attendance. Dr. Elmer Hess, Erie, president-elect, presented the society's fifty year testimonial to the honor guest, Dr. John L. Mansuy, Ralston. Among the speakers were Dr. Joseph S. Lawrence, Director, Washington Office, Council on Medical Service, A. M. A., Washington, D. C., who spoke on "Preventive Medicine: Facts and Fancies"; Dr. Howard K. Petry, Harrisburg, president, Medical Society of the State of Pennsylvania, "Role of the State and the Local Community in a Mental Health Program," and Dr. Pascal F. Lucchesi, superintendent, Philadelphia General Hospital, "The Family Doctor's Role in Public Health Preventive Medicine."

Philadelphia

Shmookler Memorial Lecture.—The H. B. Shmookler Memorial Lecture of Mount Sinai Hospital will be delivered by Dr. Louis N. Katz, Chicago, on "The Control of the Coronary Circulation," June 5, at 9 p. m. in the nurses' conference room.

Dr. Long to Head Tuberculosis and Health Association.—Dr. Esmond R. Long, director of the Henry Phipps Institute, University of Pennsylvania, has been elected president of the Philadelphia Tuberculosis and Health Association to succeed Dr. Charles J. Hatfield, president of the organization for twenty-four years. The board has made Dr. Hatfield president emeritus in recognition of his long period of service.

RHODE ISLAND

Society News.—The Rhode Island Society of Industrial Physicians and Surgeons, Inc., have elected the following officers for the coming year: Drs. Richard F. McCoart, Providence, president; Thomas A. Egan, Providence, vice president; James P. Deery, Providence, secretary, and Robert T. Henry, Pawtucket, treasurer.

TEXAS

State Medical Election.—At the annual meeting, May 8, of the State Medical Association of Texas the following officers were elected: president, Dr. Tate Miller, Dallas; vice presidents, Drs. Frank A. Selecman, Dallas; Ximie R. Hyde, Fort Worth, and Elmer W. Jones, Wellington; secretary, Dr. Holman Taylor, Fort Worth, and treasurer, Dr. Thomas H. Thomason, Fort Worth. The next annual session will be in Houston.

WEST VIRGINIA

State Medical Election.—The West Virginia State Medical Association elected the following officers at the annual meeting in Charleston May 13: president, Dr. Thomas F. Bess, Keyser; first vice president, Dr. Spencer L. Bivens, Charleston; second vice president, Dr. Harold Van Hoose, Man, and treasurer, Dr. Thomas M. Barber, Charleston. Dr. Ivan Fawcett, Wheeling, was chosen A. M. A. delegate with Dr. James L. Wade, Parkersburg, alternate. Both succeed themselves. Total doctor registration broke all state records, 559 compared with a previous high of 512 at Clarksburg in 1937. The next annual meeting will be held at the Greenbrier Hotel, White Sulphur Springs, July 12-14, 1948.

PUERTO RICO

Health Legislation in Puerto Rico.—The governor of Puerto Rico, Hon. Jesus T. Pinero, has recently approved the following appropriation bills passed by the insular legislature in its regular annual session meeting February to April, to carry out the government's health and sanitation program during the coming fiscal year:

House bill 1374, authorizing a hospital survey of the island to determine the need for additional hospital facilities.

Act 69, appropriating the sum of $150,000 to the department of health to carry on its malaria control program during the fiscal year.

Act 33, appropriating the sum of $51,000 for improvements to the tuberculosis hospital in the city of Ponce.

Act 41, appropriating the sum of $55,000 for improvements to the tuberculosis hospital in the city of Mayaguez.

Act 43, appropriating the sum of $25,000 to continue the enlargement and new constructions to municipal hospitals in the island.

Act 44, appropriating $100,000 for the acquisition, transportation and installation of latrines in the rural areas.

Act 48, appropriating $38,000 for improvement to the tuberculosis hospital in the city of Guayama.

Act 53, appropriating $187,500 to the University of Puerto Rico for scholarships for poor medical students to be sent to colleges and universities in the United States.

Act 392, providing for the construction of a municipal hospital in Piedras.

Act. 413, appropriating $75,000 for improvements and enlargement to the municipal hospitals in the city of San Juan and for purchase of equipment for these hospitals.

Act 112, appropriating $120,000 for repairs, extension and equipment of the Hospital for the Lepers, established in the city of Trujillo Alto.

Act 410, appropriating $6,250,000 to carry on a vigorous slum clearance program in Puerto Rico.

Act 437, authorizing the administrator of the State Insurance Fund to use the services of American and foreign physicians, who shall be contacted by the insular Department of Health.

Act 444, to prohibit contamination of potable water sources in Puerto Rico, to provide penalties for violation of this act and to create a commission to intervene in such cases.

Act. 370, authorizing the department of health to copy in microfilm all the birth certificates, marriage licenses and death certificates that appear in the old civil registry of Puerto Rico in order to preserve such documents, and also authorizing that microfilms be made of all such documents issued by the vital statistics bureau of the department of health to interested parties.

Act 380, providing that credit as alternates be granted to all physicians who served in hospitals during active military service in the armed forces of the United States, who will serve in government institutions in Puerto Rico.

GENERAL

Pan American Congress of Pediatrics.—The first Pan American Congress of Pediatrics will be held at the Mayflower Hotel, Washington, D. C., July 9 to 12, under the auspices of the American Academy of Pediatrics. Dr. Felix Hurtado Galtes, Havana, will be the presiding officer.

Percy Jones Hospital Reunion.—The Percy Jones Hospital reunion will be held at Longport Inn, 35th and Atlantic avenues, Longport, N. J., Monday, June 9. 7 p. m. Further information can be obtained from Mrs. Kathryn S. Read, 5407 Atlantic Avenue, Ventnor, N. J., phone Atlantic City 2-3509.

Clinical Pathologists Cancel Atlantic City Meeting.—The Atlantic City meeting of the American Society of Clinical Pathologists listed in Coming Meetings as June 4-8 has been canceled in view of the meeting of the United Nations. The society will meet in Chicago October 27-30 instead.

Appoint Director of Commonwealth Fund.—Dr. Donal Sheehan, professor of anatomy and lately acting dean of the New York University College of Medicine, New York, has been appointed general director of the Commonwealth Fund to succeed Barry C. Smith, who will retire September 1. Dr. Sheehan came to this country from England in 1937 to join the faculty of New York University. He recently completed a mission to Czechoslovakia for the Rockefeller Foundation.

Heart Association Appointment.—Dr. Charles A. R. Connor has been appointed associate medical director of the American Heart Association and will work with Dr. David D. Rutstein, medical director, in developing the association's program of research service and public education in diseases of the heart and circulation. Dr. Connor, who received his medical degree in 1931 and his Doctor of Science in Medicine from New York University College of Medicine, New York, in 1940, has been since 1935 instructor in medicine there. In 1946 he was named attending physician in cardiovascular disease, Lenox Hill Hospital, New York.

Dr. Waksman to Receive Passano Foundation Award.—At a presentation dinner scheduled for June 12 during the week of the Centennial Celebration in Atlantic City, Selman A. Waksman, Sc.D., New Brunswick, N. J., will receive the $5,000 Passano Foundation award for original research in the field of antibiotics culminating with his discovery of streptomycin. Dr. Waksman will give an address, "Antibiotics and Tuberculosis—A Microbiological Approach," and Sir Howard Florey, Ph.D., Oxford, England, who was knighted for his development of the clinical applications of penicillin, will give a brief address. The Passano Foundation, established in 1943 by the Williams & Wilkins Company, medical publishers, Baltimore, provides the award to encourage medical research.

Assistant Director of American College of Surgeons.—Dr. Charles F. Branch, formerly dean of Boston University School of Medicine and recently director of the Children's Hospital, Boston, has joined the staff of the American College of Surgeons in Chicago as assistant director. He will be particularly concerned with the work of the cancer committee of the college, which includes a survey and approval of cancer clinics inaugurated in 1930 and the establishment of a similar program in relation to cancer detection centers. Dr. Branch, a graduate of the University of Vermont College of Medicine, Burlington, was professor of pathology at Boston University School of Medicine and pathologist to the Massachusetts Memorial Hospitals as well as serving a number of suburban hospitals in Boston from 1926 to 1946. He is a past president of the New England Pathological Society.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery.—This society will meet at the Hotel Mayflower, Atlantic City, June 11, at 8 p. m. The speakers will include Dr. Norman N. Smith, New Haven, Conn., "Preparation of the Patient for Rhinoplasty"; Dr. Frank Turchik, Bridgeport, Conn., "Annoying Postoperative Problems in Nasal and Septal Plastic Surgery"; Dr. Samuel F. Kelley, New York, "Tantalum Implants for Correction of Postoperative Deformity Resulting from Radical Frontal Sinus Surgery"; Drs. Joseph G. Gilbert and Joseph S. Silverberg, Brooklyn, "Evaluation of the Various Types of Implants for Correction of Saddle Nose"; Dr. John F. Pick, associate in surgery, University of Illinois, Chicago, "Plastic Otorhinologic Surgery." General discussion will be opened by Dr. Francis W. White, New York.

Dr. Cori Receives Award.—Dr. Carl F. Cori, Washington University Medical School, St. Louis, was awarded the second intermediate research prize of $5,000 April 28 for distinguished achievements in the field of human metabolism extending over a period of twenty years. The award, made annually by the National Science Fund of the National Academy of Sciences

in recognition of the development of original knowledge about sugar, was presented by Dr. William C. Stadie, associate professor of research medicine at the University of Pennsylvania School of Medicine, Philadelphia. The most significant contribution Dr. Cori has made is indicating how insulin influences the burning of sugar, opening the way for improved technics for the control of diabetes. His research has led to the discovery of the enzymes phosphorylase and hexokinase. More recently Dr. Cori and his wife indicated the mechanism for the release of stored blood sugar in the liver.

Norton Medical Award for Book Manuscripts.—W. W. Norton & Company are again offering the Norton Medical Award for book manuscripts written for the lay public by professional workers in the field of medicine. The publishers now set no final closing date for the submission of manuscripts which may be submitted at any time, the award not being limited to any one year. The Norton Award offers $5,000 as a guaranteed advance against royalties. Either complete manuscripts or detailed table of contents together with 100 pages of manuscript may be submitted. A folder giving details of the terms of the award may be secured from the publishers at 101 Fifth Avenue, New York 3. Books that have previously won Norton Medical Awards are "The Doctor's Job," by Dr. Carl A. L. Binger, New York; "Doctors East, Doctors West," by Dr. Edward H. Hume, New York, and "A Surgeon's Domain," by Dr. Bertram M. Bernheim, Baltimore, published this spring.

Research in Tuberculosis.—At a recent meeting the committee on medical research of the National Tuberculosis Association, New York, approved the following grants for research and allocated a total of $91,550 for these purposes for the fiscal year ending March 31, 1948:

1. Analysis of internal chemistry of the tubercle bacillus, and cataloguing of the chemical derivatives.—Dr. Ralph J. Anderson, Yale University, New Haven, Conn.
2. Investigation of pulmonary calcifications.—Dr. Amos Christie, Vanderbilt University, Nashville, Tenn.
3. Synthesis of biologic reaction of different chemical fractions of tubercle bacillus provided by chemical laboratories.—Dr. Charles A. Doan, Ohio State University, Columbus, Ohio. (This study is supported by funds contributed by the Ohio Tuberculosis and Health Association.)
4. Determination of the course of tuberculosis in children exposed to the disease from infancy to adult life.—Dr. Edith M. Lincoln, Bellevue Hospital, New York.
5. Determination of the relation of tubercle bacillus carbohydrates to tuberculosis and to its immunity.—Dr. Sidney Raffel, Stanford University, Calif. (This is a cooperative study partially supported by funds contributed by the California Tuberculosis and Health Association.)
6. The chemistry of tuberculin.—Dr. Florence B. Seibert, Henry Phipps Institute, Philadelphia.
7. Provision of base line data on the serum content of different globulins and polysaccharides in normal animals used for research in tuberculosis.—Dr. J. W. Willliams, University of Wisconsin, Madison, Wis.
8. The role of endocellular proteolytic enzymes during stages of caseation and softening.—Dr. Charles Weiss, Jewish Hospital, Philadelphia.
9. Investigation of some of the enzymatic factors involved in caseation of tuberculosis.—Dr. Donald E. Bowman, University of Indiana, Indianapolis. (This study is supported by funds contributed by the Indiana Tuberculosis Association and will be correlated with the previous study [No. 8].)
10. Study of vitamin metabolism in relation to growth of human and bovine type tubercle bacilli.—Dr. David T. Smith, Duke University, Durham, N. C.
11. Pathologic study of lobes and lungs removed surgically and available for histologic investigation.—Dr. William Meissner, N. E. Deaconess Hospital, Boston.
12. Tabulation of statistical results of: (a) Pregnancy and tuberculosis, (b) tuberculosis in nurses.—Dr. T. L. Badger, Boston City Hospital, Boston.
13. Provision of standard strains of tubercle bacilli whose history, virulence and, as far as possible, chemical structure is known.—Dr. Edward N. Packard, Saranac Lake Laboratory, Saranac Lake, N. Y.
14. Investigation of forces responsible for movement of air into and out of the lungs.—Dr. George W. Wright, Edward L. Trudeau Foundation, Saranac, N. Y.
15. Study of influence of posture on lung size.—Dr. George W. Wright, Edward L. Trudeau Foundation.
16. Determiation of best laboratory procedures for culture mediums.—Mr. William Steenken, Edward L. Trudeau Foundation; Dr. C. Richard Smith, Barlow Sanatorium, Los Angeles; Dr. M. I. Smith, National Institute of Health, U. S. Public Health Service, Bethesda, Md.
17. Study of minimal tuberculosis in nurses.—Dr. Carroll E. Palmer, National Institute of Health, Bethesda, Md.
18. Studies for the determination of the effectiveness of streptomycin in the treatment of tuberculosis.—A series of projects in cooperation with the National Institute of Health, Bethesda, Md.

NOTE: The following tuberculosis associations have contributed funds to the medical research program but have left the decision for their allocation to the National Tuberculosis Association: Pennsylvania Tuberculosis Society, Philadelphia, Philadelphia Tuberculosis and Health Association, Philadelphia, and Reading Tuberculosis Association, Reading, Pa.

Appoint Associate Director of National Health Council.—Dr. John W. Ferree, New York, formerly director, division of educational services, American Social Hygiene Association, has been appointed executive director of the National Health Council. He will be engaged principally in helping existing local, county and state health councils broaden the scope of their usefulness and in stimulating the formation of councils where their establishment would advance the effectiveness of community and state health agencies. Dr. Ferree received his medical degree from the Indiana University School of Medicine, Bloomington, and his Master of Public Health degree from Johns Hopkins University School of Public Health and Hygiene, Baltimore. He was chief of the bureau of local health administration, Indiana State Board of Health, 1936-1938, and from 1940 to 1942 he was state health commissioner for Indiana. After retiring from active duty with the Medical Corps, U. S. Navy, in 1945 with the rank of commander, Dr. Ferree joined the staff of the American Social Hygiene Association.

American Diabetes Association Meeting.—The annual meeting of the American Diabetes Association will be held, June 7-8, at the Chalfonte-Haddon Hall, Atlantic City, under the presidency of Dr. Russell M. Wilder, Rochester, Minn. Speakers on the program at 2 p. m. include Dr. Joseph Berkson, Mayo Clinic, Rochester, "Life Expectancy in Diabetes"; Drs. E. T. Waters, associate professor of physiology, and Charles H. Best, University of Toronto, "Importance of Developing an Accurate Method for Estimation of Insulin in the Blood"; Dr. Hugh L. C. Wilkerson, Boston, "Office of Diabetes, U. S. Public Health Service, Plans and Program"; Drs. Ferdinand R. Schemm, Felix H. Crago and John A. Layne, Great Falls (Montana) Clinic, "Control of Edema in Diabetic Patients with General Vascular Disease," and Dr. Geza de Takats, University of Illinois College of Medicine, Chicago, "Delayed Primary Suture in the Surgical Management of Diabetic Gangrene." At the banquet Sunday night the Banting Lecture will be delivered by Dr. George H. A. Clowes, Indianapolis, on "Development of Large Scale Production of Insulin."

CANADA

Survey of Milk Supplies.—In a recent survey in the eight provinces outside Ontario it was revealed that of the 366 municipalities represented only 13 per cent have compulsory pasteurization by-laws. Ontario was excluded because it is the only province which has a province-wide law in force. Of cities and towns in Saskatchewan 20 per cent reported safe milk by-laws; Quebec was second with 15 per cent; ninety municipalities, or 20 per cent, reported that their supply was unpasteurized; sixteen revealed that while they had no compulsory law their supply was 100 per cent pasteurized. Forty-one municipalities revealed that 90 per cent or more of their milk supply was pasteurized.

The Annual Meeting.—The Canadian Medical Association will hold its annual meeting June 23-27 in the Royal Alexandria Hotel, Winnipeg, under the presidency of Dr. Wallace Wilson, Vancouver. The program will consist of round table conferences to be held the mornings of June 25-27 and sectional meetings to be held the afternoons of those days. A panel discussion on the provision for medical care is being arranged for Thursday evening, June 26, by the committee on medical economics. Among the subjects of the round table conferences will be Management of Hypertension, Cesarean Section—Indications and Contraindications, and Management of Bulbar Poliomyelitis. Among the speakers will be Drs. Edgar J. Huenekens, Robert E. Priest and Abe B. Baker, all of Minneapolis. On Wednesday the Osler Lecture will be presented by Professor John M. McMichael, London, England, on "Osler: The Textbook and Medical Education." Among the speakers at the section meetings will be Dr. Paul A. O'Leary, Rochester, Minn., on "Recent Concepts of the Dermatoscleroses" in the section of dermatology; Dr. Warren H. Cole, Chicago, whose subject will be announced, in the section of surgery; Dr. Leo G. Rigler, Minneapolis, "Emphysema: An Early Sign of Bronchogenic Carcinoma" in the section of radiology, and Dr. E. Bruce Tovee, Chicago, "Present Status of Vagus Section in Ulceration of the Stomach and Duodenum" in the section of surgery.

Marriages

HAROLD H. KERR, Akron, Iowa, to Miss Norma Hayes Trabold of Wrightstown, Pa., in New York February 15.

WALTER K. HOFFMAN, Memphis, Tenn., to Mrs. Phyllis Benson Halladay of Oakland, Calif., January 25.

STEPHEN ABRAM GREENBERG, Florence, S. C., to Miss Ruth Barbara Brody in New York January 26.

BIRT L. BROWNING, Miami Beach, Fla., to Mrs. Bessie Hickman in Brunswick, Ga., Dec. 24, 1946.

JUNE U. GUNTER, Sanford, N. C., to Miss Elizabeth G. Kerr of Philadelphia February 1.

Deaths

Henry Hamilton Moore Lyle ✠ New York; born in Connor County, Ulster Province, Ireland, Nov. 13, 1875; Columbia University College of Physicians and Surgeons, New York, 1900; professor of clinical surgery at the Cornell University Medical College; member of the American Surgical Association and the Society of Clinical Surgery; fellow of the American College of Surgeons; served overseas during World War I and won the American Distinguished Service Medal, the British War Medal, the British Victory Medal and other decorations; consulting surgeon, Stuyvesant Square Hospital and Veterans Administration, district number 2, in New York, Elizabeth A. Horton Memorial Hospital in Middletown, Cornwall Hospital in Cornwall, New York State Reconstruction Home for Children in West Haverstraw, St. Luke's Hospital in Newburgh and St. Luke's Hospital in New York, where he died March 11, aged 71, of coronary heart disease.

Lois Ellene Taylor, Belchertown, Mass.; born in Poultney, Vt., July 8, 1909; University of Vermont College of Medicine, Burlington, 1933; interned at the New England Hospital for Women and Children in Boston; served a residency at the Danvers State Hospital in Danvers and the Brattleboro Retreat in Brattleboro, Vt.; diplomate of the National Board of Medical Examiners; member of the New England Society of Psychiatry and the American Medical Association; lieutenant, senior grade, in the U. S. Naval Reserve during World War II; winner of the Sesquicentennial American Youth Award for 1926 and was presented with a gold medal by President Calvin Coolidge; since 1937 senior physician at the Belchertown State Hospital, where she died March 21, aged 37.

James E. King ✠ Buffalo; born in Buffalo, Sept. 15, 1874; University of Buffalo School of Medicine, 1896; professor of gynecology emeritus at his alma mater; specialist certified by the American Board of Obstetrics and Gynecology; member and past president of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons; past president of the Buffalo Academy of Medicine, Medical Society of Erie County and the University of Buffalo Alumni Association; fellow and formerly vice president of the American Gynecological Society; fellow of the International College of Surgeons and the American College of Surgeons; served on the staffs of the Buffalo General and the Edward J. Meyer Memorial hospitals; died March 9, aged 72, of heart disease.

William Sharp Lindsay, Topeka, Kan.; College of Physicians and Surgeons, Keokuk, Iowa, 1874; member of the American Medical Association and in 1919 and 1920 member of the House of Delegates; served as president of the Kansas State Medical Society and the Shawnee County Medical Society; formerly dean and professor at the Kansas Medical College; at one time associated with the Topeka State Hospital, where he served as superintendent; formerly in charge of the Cottage for Mental and Nervous Diseases, Christ's Hospital; for many years trustee of the Washburn College; died February 20, aged 94, of heart disease.

Frank Edward Artaud ✠ Major, U. S. Army, retired, Treasure Island, Fla.; Medical Department of Tulane University of Louisiana, New Orleans, 1884; member of the Medical Association of Georgia; veteran of the Spanish-American War and World War I; entered the regular army medical corps on March 15, 1927 as a first lieutenant; retired March 15, 1927 for disability in line of duty; promoted to major under the special retirement act of Congress for June 21, 1930; died in the Veterans Administration Hospital, Bay Pines, April 9, aged 87 of coronary sclerosis.

Wilbur Arthur Baker ✠ Kansas City, Mo.; University of Kansas School of Medicine, Kansas City, 1917; on the courtesy staff of the Menorah Hospital; died in Phoenix, Ariz., February 24, aged 54, of coronary thrombosis.

George Alexander Brandon, Jackson, Tenn.; University of Nashville Medical Department, 1906; member of the American Medical Association; affiliated with the Henderson County draft boards during World War I and World War II; died February 26, aged 72, of cerebral hemorrhage.

Jerry B. Callahan, Orlando, Fla.; Leonard Medical School, Raleigh, 1907; died February 28, aged 63, of coronary heart disease.

Robert Taylor Childress, Kingsport, Tenn. (licensed in Tennessee in 1911); served as chairman of the county school board; died February 22, aged 68, of hypertension.

Chester Cyril Coates, New York; Illinois Medical College, Chicago, 1899; died in St. Clare's Hospital February 10, aged 73.

Joseph Evan Crawford ✠ Ruston, La.; Memphis (Tenn.) Hospital Medical College, 1907; on the staff of the Ruston-Lincoln Sanitarium, where he died February 17, aged 68, of cerebral hemorrhage.

George Byron Cross, Lakeville, Minn.; Chicago Homeopathic Medical College, 1901; died recently, aged 68, of cerebral thrombosis.

Frank C. Dildine, Port Jefferson, N. Y.; Baltimore Medical College, 1897; member of the American Medical Association; died February 17, aged 79, of accidental monoxide gas poisoning.

Theodore David Dockstader, Ravena, N. Y.; Albany Medical College, 1905; died February 13, aged 63, of multiple sclerosis.

Pressly Young Donald, Selma, Ala.; Tulane University of Louisiana School of Medicine, New Orleans, 1915; member of the American Medical Association and the Southeastern Surgical Congress; past president of the Dallas County Medical Society; served during World War I; member of the city school board; chairman of the county board of health; on the staffs of the Good Samaritan Hospital and King Memorial Hospital, where he died February 26, aged 57, of acute gangrenous cholecystitis and cholelithiasis.

Joseph Koger Fairey, St. Matthews, S. C.; Medical College of the State of South Carolina, Charleston, 1891; member of the American Medical Association; died February 21, aged 78, of pneumonia.

David Ferguson Jr., ✠ Medical Inspector, Commander, U. S. Navy, retired, Milledgeville, Ga.; Emory University School of Medicine, Atlanta, 1914; specialist certified by the American Board of Internal Medicine; entered the U. S. Navy on Dec. 31, 1917 and retired Dec. 1, 1941 for incapacity resulting from an incident of the service; died Aug. 19, 1946, aged 53.

Emma Culross Gibbons, Portland, Maine; McGill University Faculty of Medicine, Montreal, Que., Canada, 1923; died February 16, aged 71, of coronary occlusion.

Vincent Jones Gragg ✠ Magnolia Springs, Ala.; Medical Department of Tulane University of Louisiana, New Orleans, 1908; for many years health officer of Chilton County; at one time mayor of Clanton; president of the Baldwin County Medical Society; member of the board of directors and served as president of the Central Alabama Hospital in Clanton; died in the Highland Avenue Baptist Hospital, Birmingham, February 17, aged 70, of intestinal obstruction.

Edwin Austin Hatch, Brooklyn; Long Island College Hospital, Brooklyn, 1881; member of the American Medical Association; formerly on the staff of St. Mary's Hospital; died recently, aged 91, of ruptured aneurysm and arteriosclerosis.

Jacob Roland Heller, Allentown, Pa.; University of Pennsylvania School of Medicine, Philadelphia, 1928; member of the American Medical Association; medical examiner for the school district in Allentown; interned at the Allentown Hospital, where he served as a member of the staff; began active duty as a lieutenant commander in the U. S. Naval Reserve in October 1942; service terminated in March 1946 with the rank of commander; died February 2, aged 44, of coronary occlusion.

David Williams Henderson ✠ Salt Lake City; Jefferson Medical College of Philadelphia, 1912; specialist certified by the American Board of Ophthalmology; member of the Western Ophthalmological Society and the Pacific Coast Oto-Ophthalmological Society; fellow of the American College of Surgeons; member of the staff of the Dr. W. H. Groves Latter Day Saints Hospital; died February 1, aged 65, of carcinoma.

Frederick Kingsley ✠ Boston; Queen's University Faculty of Medicine, Kingston, Ont., Canada, 1905; for many years consultant at the Medfield (Mass.) State Hospital; died February 1, aged 66, of coronary thrombosis

John Raymond Lynch, New York; Fordham University School of Medicine, New York, 1915; member of the American Medical Association; served during World War I; died in the Fitch Sanitarium January 21, aged 54.

Jesse L. Macbeth ✠ St. Clair, Minn.; Fort Wayne College of Medicine, 1905; died in St. Joseph's Hospital in Mankato recently, aged 66, of carcinoma.

John Tate McNabb, Trenton, Tenn.; University of Tennessee College of Medicine, Memphis, 1934; interned at the Baroness Erlanger Hospital, Pine Breeze Sanatorium and the Children's hospitals in Chattanooga; at one time assistant physician at the Eastern State Hospital in Knoxville; member of the American Medical Association; served during World War II; formerly associated with the U. S. Public Health Service Reserve; died in Monroe, La., April 9, 1946, aged 39, of chronic myocarditis while en route to Alexandria, La., to establish his practice.

M. Mozart Marrin, Grand Rapids, Mich.; University of Michigan Medical School, Ann Arbor, 1932; member of the American Medical Association; awarded the Edward and Susan Lowe medical fellowship for 1941; fellow of the American College of Surgeons; served during World War II; on the staff of the Butterworth Hospital, where he served an internship and where he died February 8, aged 40, of coronary thrombosis.

Joseph Alston Maxwell ✠ Tuscaloosa, Ala.; Medical Department of Tulane University of Louisiana, New Orleans, 1912; served during World War I; fellow of the American College of Surgeons; on the staffs of the Druid City and Stillman hospitals; died February 14, aged 59, of heart disease.

George Henry Musso, Lynn, Mass.; New York University Medical College, 1898; member of the American Medical Association; affiliated with the Union Hospital, where he died March 1, aged 72, of rupture of the aorta.

Louis Myer Piatt ✠ Columbus, Ohio; Ohio State University College of Medicine, Columbus, 1923; specialist certified by the American Board of Radiology; affiliated with the Grant, St. Francis and Mercy hospitals; killed in an automobile accident March 2, aged 47, while returning from vacationing in Mexico.

Arnold Frederick Plankers, St. Paul; John A. Creighton Medical College, Omaha, 1910; died in the Good Samaritan Hospital recently, aged 61, of coronary thrombosis and myocarditis.

Nathan Shapiro, Brooklyn; University of Edinburgh Faculty of Medicine, Scotland, 1930; member of the American Medical Association; on the staffs of the Coney Island and Israel Zion hospitals; died recently, aged 45.

Frank Ernest Sohler ✠ Healdsburg, Calif.; Cooper Medical College, San Francisco, 1906; formerly on the staff of the French Hospital in San Francisco; died recently, aged 65.

William Joseph Topmoeller, Cincinnati; University of Cincinnati College of Medicine, 1912; cited for bravery and received the Purple Heart for services during World War I; affiliated with the Good Samaritan, Deaconess and Children's hospitals; past president of the Xavier College Alumni Association; died in the Holmes Hospital March 10, aged 57, of brain tumor.

Frank White Whitmore, St. Paul; Northwestern University Medical School, Chicago, 1915; member of the American Medical Association; clinical associate professor of nervous and mental diseases at the University of Minnesota Medical School; on the staffs of the Miller, St. Luke's, Ancker and Bethesda hospitals; died recently, aged 56, of aortic stenosis.

William Walter Wolf, Minneapolis; Hahnemann Medical College and Hospital of Philadelphia, 1939; member of the American Medical Association; served during World War II; affiliated with the Swedish and Deaconess hospitals; died Dec. 14, 1946, aged 35, of coronary thrombosis.

Lieut. Harold A. Kepnes
(MC), U. S. N. R., 1911-1942

Major Philip Bress,
M. C., A. U. S., 1908-1944

Major Carlo J. Marinello,
M. C., A. U. S., 1915-1944

Alfred Scheyer, New York; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, 1896; member of the American Medical Association; on the staffs of the Lutheran and Gotham hospitals; staff member, Westside Y. M. C. A.; died March 17, aged 73, of heart disease.

Stamatis Demosthenes Zaph ✠ Los Angeles; Chicago College of Medicine and Surgery, 1917; member of the Illinois State Medical Society; formerly affiliated with the Woodlawn Hospital in Chicago; died in Chicago March 7, aged 63, of pulmonary thrombosis.

KILLED IN ACTION

Harold Arthur Kepnes, Chelsea, Mass.; Tufts College Medical School, Boston, 1939; diplomate of the National Board of Medical Examiners; interned at the Queens General Hospital in Jamaica, N. Y.; began active duty as a lieutenant (jg) in the medical corps of the U. S. Naval Reserve on July 7, 1941; promoted to lieutenant; served with the First Marine Division; killed in action in Guadalcanal Nov. 3, 1942, aged 31.

Philip Bress, Jackson, Ky.; University of Virginia Department of Medicine, Charlottesville, 1931; member of the American Medical Association; interned at the Baltimore City Hospitals; served a residency at St. Joseph's Hospital in Baltimore, Methodist Episcopal Hospital in Fort Wayne, Ind., and the Mercy Hospital in Logan, W. Va.; major, medical corps, Army of the United States; prisoner of war of the Japanese; killed in the South China Sea Oct. 24, 1944, aged 36, when the ship on which he was being transported was sunk.

Carlo James Marinello, Buffalo; University of Buffalo School of Medicine, 1939; diplomate of the National Board of Medical Examiners; served an internship and residency at the Edward J. Meyer Memorial Hospital; entered the medical corps of the Army of the United States as a first lieutenant in October 1940; spent fourteen months in the Panama Canal Zone; promoted to major; organized the 52d Portable Surgical Hospital, serving as commanding officer; with General MacArthur in the initial invasion of the Philippines; wounded Dec. 24, 1944; died in Leyte, Philippine Islands, Dec. 31, 1944, aged 29.

Foreign Letters

LONDON

(From Our Regular Correspondent)

April 26, 1947.

Medical History in Postage Stamps

An exhibition illustrative of medical history in postage stamps, which appears to be the first of its kind in this country, was held at the Historical Section of the Royal Society of Medicine. It was accompanied with a demonstration on the epidiascope and a running commentary. Stamps of medical interest are of comparatively recent issue but have multiplied in the last twenty years. They can be divided into two classes: those bearing the portraits of medical men and those depicting medical scenes, such as hospitals and medical congresses. The first medical man to be depicted appears to have been F. Espejo of Ecuador in 1899. But the first man of eminence so honored was Boerhaave, whom the Netherlands commemorated in 1928, though France began a series in honor of Pasteur's work in 1923. Among other famous men commemorated are Vesalius, Paré, Haller, Semmelweis, Purkinje, Ramon y Cajal and Reed. Britain has been backward in this form of commemoration. Not until Sir Wilfred Grenfell was commemorated on a Newfoundland stamp in 1941 was any British medical man depicted.

Intelligence and Fertility: Are We Degenerating?

At the request of the Royal Commission on Population Sir Cyril Burt, professor of psychology, University College, London, has surveyed the evidence on the relation between fertility and intelligence, and has come to a most disquieting conclusion. In different sections of the community the birth rate shows wide differences. Using data obtained from surveys of the school population by means of intelligence tests, Burt has considered whether differences in the birth rate are likely to alter the inherited mental qualities of the nation. He has found that children of the poorer social classes have an intelligence nearly two years below that of children of the better social classes, while at the same time they are drawn from families nearly twice as large. More important than this is his conclusion that the average level of intelligence in the general population is declining. He claims that the decline during the past generation has amounted to about three or four "natal months" (meaning that the intelligence tests show that the children have reached only the level of those younger by that period). The prolongation of such a decline would gravely affect in time the mental status of the population. Thus, if the rate assumed continues, in a little over fifty years the number of pupils of scholarship ability would be approximately halved and the number of feebleminded would be doubled.

Another point made by Burt is that in the numerous working class it is the more intelligent families who contribute fewest to the next generation. In general the larger the family the lower the level of intelligence; the smaller, the more intelligent the parents. These conclusions are so important that the need for further investigation is suggested. They are certainly open to some question. An offset to the higher birth rate of the less intelligent is their higher death rate. It is well known that throughout the living world the lower the organism the greater the powers of multiplication, but that has never meant the swamping of the higher forms of life by the lower, for there are countervailing forces, and so evolution has not been prevented. Herbert Spencer long ago emphasized as fundamental the antagonism between individuation and genesis. "Every higher degree of individual evolution is followed by a lower degree of race multiplication, and vice versa."

Fatal Failure to Remove a Tourniquet

An unusual case of negligence is reported as having occurred in the hospital of the large Lancashire manufacturing town of Oldham. A man aged 53, a head carder in a cotton mill, was admitted for a plastic bone operation, which consisted in removal of a piece of bone from the right leg for grafting on the right wrist. The operation was duly performed but he was returned to bed without removal of a tourniquet which had been placed on the leg. This was allowed to remain on eleven hours, although he was in great agony and complained to the nurse that there was something tight on his leg. His complaint was not investigated until he became worse. His condition became so critical that blood transfusion was performed. He collapsed and died.

His widow entered suit for damages against the hospital and the two surgeons who performed the operation. In court their lawyer said that he admitted the liability of the defendants and that it remained only for the judge to assess damages. The judge awarded $16,000 damages and costs against the hospital and the two surgeons who performed the operation. He said that he would not comment on the sorrow which had been caused, but it was a little surprising that the tourniquet had been left on. So far no explanation has been given of this apparently unprecedented mistake. But it may be surmised that it is not unconnected with two phenomena of the postwar period—overworked surgeons and shortage of nurses involving the placing of the partially trained in too responsible positions.

ITALY

(From Our Regular Correspondent)

April 26, 1947.

Center for Studies on Antibiotics

A center for information and studies on antibiotics has been established in Milan; Professor Alexander Fleming was nominated honorary president at the opening session. Professor Carlinfanti will act as director, with Dr. G. Pauletta as technical secretary. It is the aim of the center to support and to coordinate the forces of Italian research in the field of antibiotics. For the achievement of this aim the center will promote research on antibiotic substances by supporting Italian biologists, clinicians and chemists in carrying out and publishing reports of their work and by furnishing current literature, coordinate antibiotic research in Italy by favoring collaboration between research workers, by advice concerning methods of study, by furnishing research material and by the distribution of prizes. The center will answer questions of medical practitioners concerning antibiotics, organize lectures and conferences, publish the transactions and inform other countries of the contributions of Italian scholars to the subject. The Centro Italiano Informazioni Studi Antibiotici (Italian Information Center on Antibiotic Studies) is located at Corso Concordia No. 9, Milan.

International Congress on Reproduction

The first international congress on physiopathology of reproduction and of artificial fecundation will be held in Milan in April 1948 on the initiative of the Experimental Institute "Lazzaro Spallanzani" with the support of the University of Milan. The scientific objects of the institute are of interest to all countries of the world. The preparatory committee of the congress consists of biologists, geneticists, pathologists, gynecologists and zootechnicians and of representatives of various governmental departments. The rector of the University of Milan has been nominated as honorary president. The secretary general of the congress has his headquarters near the Institute Lazzaro Spallanzani, Via Bonzetti 17, Milan.

Association of Volunteer Blood Donors

The council of directors of the National Association of Volunteer Blood Donors, at a meeting in Bergamo, discussed the democratization of the former community organizations and establishment of new associations in places which have been deprived of an efficient transfusion service, provisions for safeguarding donors, provisions in the field of assistance, insurance and food supply, and suggestions for sanctions in order to prevent abuses. The council has asked the government for urgent recognition of the association to give more authority to the association and to improve the position of the volunteer blood donors.

Convention of Gastroenterologists

At the convention in Florence of practitioners specializing in diseases of the stomach and intestine those attending favored expanding the Italian Association of Gastroenterologists to include the problems of nutrition. At the meeting in Bologna in the spring reports will be presented on megaesophagus by Pieri-Bonadies and by Basso and on duodenitis, with the exception of duodenal ulcers, by Gasbarrini and by Di Gaetano.

OSLO

(From Our Regular Correspondent)

April 26, 1947.

Methyl Alcohol Poisoning

During the war several outbreaks of methyl alcohol (methanol) poisoning occurred in persons endowed with greater thirst than discrimination in the choice of their drinks. The patients were rushed to hospitals in or near Oslo, where they came under the enterprising observation of Dr. Oluf Röe, a young ophthalmologist. He has just published a 253 page study of the subject, on which he throws light calculated to promote research on new lines. Certain earlier teachings have hinged on experiments on animals, but Röe came to the conclusion that a fundamental difference probably exists between man and beast in this respect. His material consists of 82 patients treated in seven different hospitals. As many as 48 patients suffered from severe acidosis on admission to the hospital, and Röe has found that, whatever the reaction of experimental animals, in human beings at any rate the outcome of methanol poisoning depends entirely on the degree of acidosis. Hence his advocacy of bicarbonate treatment. His studies of the antecedents of his patients have led to the conclusion that ethyl alcohol counteracts methyl alcohol, a low concentration of the former in the blood being sufficient to prevent acidosis even when the concentration of the latter is high. Röe reviews (and dismisses as unconvincing) the hitherto commonly accepted notion that individual predisposition plays an important part in methanol poisoning.

The Prophylaxis of Dental Caries

It was only by chance that your correspondent found and was then intrigued by a communication by Professor G. Toverud on the prophylaxis of dental caries in the *Odontologisk Tidskrift* (1946, No. 3). For here, in only eighteen pages packed with tables and charts, is a comprehensive review of the recent research by Professor Toverud and his staff. Some of this research was started in 1936 and was voluntary; some depended on war conditions and was therefore more or less involuntary. It is suggested that all the research workers in this field should get together and study the behavior of dental caries during World War II—one of the greatest experiments in nutrition in the history of mankind. To those who are tempted to explain dental caries as an easily understood deficiency disease preventable and curable by the dietary of our primitive ancestors, Professor Toverud addresses a timely warning. In his opinion this disease is the outcome of the complex interplay of many factors about which we have still much to learn.

BRAZIL

(From Our Regular Correspondent)

Rio de Janeiro, April 26, 1947.

Active Rheumatic Carditis in Cardiac Failure

Rheumatism is considered in Brazil a relatively minor factor in the etiology of heart lesions, owing to the warm climate and the high incidence of syphilis in the population. Dr. Jairo Ramos, professor at the Paulista School of Medicine, São Paulo, has published a paper on cardiac failure in young patients through the involvement of the myocardium by an active rheumatic process. The report includes the analysis of 400 cases of acute and chronic cardiac rheumatism, among which 89 cases of rheumatic carditis were discovered (22.25 per cent). The criterion for the clinical diagnosis was to consider as rheumatic carditis the cases presenting simultaneously the largest number of signs of active rheumatism, particularly when the age was over 30 years. The age distribution in the 89 cases of active rheumatic carditis was 0-9 years 22 cases, 10-19 years 43, 20-29 years 17, 30-39 years 6, 40-49 years 1. The incidence of active rheumatic carditis within each ten year age group among the 400 cases of cardiac rheumatism varied: 0-9 years 22 out of 33, 10-19 years 43 out of 104, 20-29 years 17 out of 88, 30-39 years 6 out of 88, 40-49 years 1 out of 53, 50-59 years 0 out of 21. According to these figures the highest incidence of active rheumatic carditis occurs in young patients, a fact which is in accordance with the data in the medical literature of Europe and the United States. Below 20 years of age more than half the patients with cardiac rheumatism present clinically active rheumatic carditis; above 40 years the condition is practically nonexistent.

Influence of Variation of Cellular Permeability on Immunity

Dr. F. da Rocha Lagoa of the Oswaldo Cruz Institute, Rio de Janeiro, recently completed studies on the influence of the variation of cellular permeability on the organic resistance to infections. He used three groups of substances. The first group comprised substances that increase the permeability—testosterone, acetylcholine and the diffusion factor of staphylococcus as demonstrated by Viale, McClean, Okamoto, Gellhorn and Duran-Reynals. The second group comprised substances that decrease the cellular permeability—atropine, epinephrine and calcium as demonstrated by Okamoto, Gellhorn and Voloskov. The third group was represented by adrenal cortex extract, which maintains without variation the cellular permeability, as recently demonstrated by Rauschschwalbe and by Cicardo. The author used synthetic testosterone propionate, acetylcholine Roche and Eschatin Parke-Davis. To ascertain the resistance to infections, several hundred adult mice were infected with sufficient amounts of Klebsiella pneumoniae, Pseudomonas aeruginosa, Salmonella enteritidis and Diplococcus pneumoniae. Adult rabbits were used for the determination of the production of antibodies (hemolysins, precipitins and agglutinins).

His main conclusions are that cellular permeability plays an important part in the development of infections. Substances that increase the permeability contribute to the development of infections; substances that decrease the permeability have no disclosed influence on the course of infections; substances which contribute to maintain the normal cellular permeability increase the resistance to infections. The substances that have been studied as modifying or not the cellular permeability do not exercise any influence worth mentioning on the formation of hemolysins, agglutinins and precipitins.

Correspondence

FATAL ALLERGIC REACTION DUE TO INFLUENZA VACCINE

To the Editor:—In the article with this title by Dr. Theodore J. Curphey in THE JOURNAL, April 12, the death described was, in my opinion, not due to the egg but to sensitivity to the bacteria of the vaccine. Egg allergies in children produce definite skin or other evidences of sensitivities with which parents are familiar and which they delete from their diets. Even a positive skin test would not definitely establish the cause as due to egg, because this patient had been having egg without symptoms.

In favor of bacterial allergy is the history of aspirin sensitivity, which, in my experience, is frequently a concomitant of bacterial sensitivity. I have seen small doses of catarrhal vaccine produce symptoms approaching the description of this fatal case, and for this reason we commence all our vaccine therapy with dilutions. For example, a supervising nurse at one of our allergy clinics was started with a dilution of 1 : 1,000,000, 0.02 cc., because a 1 : 10 dilution originally produced a violent constitutional symptom. A disabled policeman with asthma almost died with symptoms similar to those described in Dr. Curphey's case from a small dose of catarrhal vaccine; he too was sensitive to aspirin.

Although an intracutaneous test for egg sensitivity may throw some light on a case within twenty minutes, bacterial allergy cannot be diagnosed by skin tests alone.

The answer to the problem may be eased by not giving influenza vaccine in one dose. Three or more ascending doses are advisable; if there is a history of aspirin sensitivity the vaccine should not be used, but if it appears necessary epinephrine hydrochloride 1 : 1,000 should be injected twenty minutes before and an ephedrine phenobarbital combination should be given every four hours after the injection for at least three days (Serum Administration and Allergy, Queries and Minor Notes, THE JOURNAL, May 20, 1944, p. 238).

DAVID LOUIS ENGELSHER, M.D., New York.

ECLAMPSIA

To the Editor:—In connection with the "Contribution to the Study of Eclampsia" by Whitacre, Loeb and Chin in THE JOURNAL, February 15, may I draw your attention to the publication "Protein Depletion in Pregnancy Toxemia" in the *Western Journal of Surgery, Obstetric and Gynecology* (**54:** 392 [Oct.] 1946). The main sequence in eclampsia is (1) fetal demand for maternal protein, aggravated by inadequate protein intake, (2) hypoalbuminemia, (3) hypoproteinemic cerebral edema, (4) increased intracranial pressure and (5) elevation of blood pressure. There are many side effects complicating the picture. Changes in the kidneys are secondary and may be partly responsible for the high blood pressure. The important cause of the elevation of blood pressure in eclampsia is the increase in intracranial pressure. Hypoalbuminemia is a constant finding in pregnancy toxemia.

H. SPITZER, M.D., Curaçao, NWI.

HEPATIC IMPAIRMENT

To the Editor:—A number of questions arise when one reads the article by Mateer and his associates on hepatic impairment in THE JOURNAL, March 29. Their use of screening liver function tests in over 300 healthy individuals resulted in designating about 20 per cent as cases of "subclinical liver impairment." In the absence of exposure to known toxins or infectious agents this percentage appears high. Assuming that this group contains no false positives, what proportion may be expected to emerge as clinical hepatopathies? Has a control group of patients positive to liver function tests who have not had a therapeutic test been followed? What percentage of these show subsequent negative function tests?

On the other hand, in the presence of so many false negatives both in clinical and in subclinical liver disease, may not many so-called subclinical cases be false positives? The intangibles involved in studying a subclinical disease entity must be tremendous, especially when utilizing laboratory tests whose exact mechanisms are not well understood.

SIDNEY COHEN, M.D.,
Wadsworth General Hospital,
Los Angeles.

TRANSFUSION MALARIA

To the Editor:—I was interested in reading the letter from Dr. Coggeshall on the danger of transfusion malaria which appeared in the April 19 issue of THE JOURNAL. It is well known, of course, that there is a risk of malaria developing in the recipient when blood is used from a donor who has had malaria within the past few or several years, even though the blood may have been stored in a blood bank for a period of days. However, I believe one should not overemphasize the danger of transfusion malaria, because it is easily controlled, and one should not hesitate to use, in the face of an emergency, a donor who has had malaria even recently. In such a case, either the donor or the recipient should be treated with atabrine or chloroquin, which would most certainly prevent the development of malaria in the recipient. Furthermore, transfusion malaria, being trophozoite induced, does not give rise to "exoerythrocytic forms." Hence, relapses do not occur.

GEORGE M. SAUNDERS, M.D., St. Louis.
Assistant Professor of Preventive Medicine, Washington University School of Medicine.

"MUMPSIMUS"

To the Editor:—I noticed that in THE JOURNAL, Dec. 14, 1946, you commented on John Forbes's article Myth and Mumpsimus and gave a meaningless explanation (though correct) of the derivation of mumpsimus.

To give the person, tense and mood of the verb *sumo*, from which sumpsimus was corrupted, gives no clue to the meaning whatsoever.

The explanation is that an illiterate English priest, when corrected for reading "Quod in ore mumpsimus" in The Mass, replied "I will not change my old mumpsimus for your new sumpsimus." This story originally came out in R. Pace De Fructu 1517, page 80—but maybe you have not had access to this reference. You can check this in the New English Dictionary or Ivor Brown's book "Just Another Word."

RICHARD ASHER, M.D., M.R.C.P.
2, Seaford Court,
Great Portland Street,
London, W. 1., England.

DISSOLUTION OF TEETH BY LEMON JUICE

To the Editor:—The editorial comment on "Dissolution of Substance of Teeth by Lemon Juice" in the March 19 issue of THE JOURNAL is of great interest. However, it should be noted that this observation was first made by S. C. Miller of New York University College of Dentistry in 1935 and commented on in an editorial in *Dental Items of Interest* in October 1935. It is also discussed in Dr. Miller's Oral Diagnosis and Treatment (Philadelphia, Blakiston Company, 1936, p. 263; 1946, p. 367) and his Textbook of Periodontia (1938, p. 433; 1943, p. 61).

MORRIS WARREN, D.D.S., New York.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

NATIONAL BOARD OF MEDICAL EXAMINERS EXAMINING BOARDS IN SPECIALTIES

Examinations of the National Board of Medical Examiners and the Examining Boards in Specialties were published in THE JOURNAL, May 24, page 395.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: Montgomery, June 24-26. Sec., Dr. D. G. Gill, 519 Dexter Ave., Montgomery 4.

ARIZONA: * *Examination.* Phoenix, July 1-2. Sec., Dr. J. H. Patterson, 826 Security Bldg., Phoenix.

ARKANSAS: * *Medical. Examination.* June 5-6. Sec., Medical Board of the Arkansas Medical Society, Dr. L. J. Kosminsky, Texarkana. *Eclectic.* Little Rock, June 5. Sec., Dr. C. H. Young, 1415 Main St., Little Rock.

CALIFORNIA: *Written.* San Francisco, July 7-10. *Oral.* San Francisco, July 19-20. *Oral Clinical Examination for Foreign Medical Graduates.* San Francisco, June 21. Sec., Dr. Frederick N. Scatena, 1020 N. St., Sacramento 14.

COLORADO: * Denver, July 18. Final date for filing application is June 15. Sec., Dr. J. B. Davis, 831 Republic Bldg., Denver.

CONNECTICUT: *Examination.* Hartford, July 8-9. Sec., Dr. Creighton Barker, 258 Church St., New Haven. *Homeopathic.* Derby, July 8. Sec., Dr. Joseph H. Evans, 1488 Chapel St., New Haven.

DELAWARE: *Examination.* Dover, July 8-10. *Endorsement.* Dover, July 15. Sec., Medical Council of Delaware, Dr. J. S. McDaniel, 229 S. State St., Dover.

DISTRICT OF COLUMBIA: * *Reciprocity.* Washington, June 9. Sec., Commission on Licensure, Dr. George C. Ruhland, 6012 E. Municipal Bldg., Washington 1.

FLORIDA: * *Examination.* Jacksonville, June 24-25. Sec., Dr. Harold D. Van Schaick, 2736 S. W. Seventh Ave., Miami 36.

GEORGIA: Atlanta, Oct. 14-16. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

HAWAII: *Examination.* Honolulu, July 8-11. Sec., Dr. S. E. Doolittle, 881 S. Hotel St., Honolulu 53.

IDAHO: Boise, July 14-16. Sec., Bureau of Occupational Licenses, Dr. S. M. Poindexter, Eastman Bldg., Boise.

ILLINOIS: Chicago, June 24-26. Sec., Mr. Philip M. Harman, Supt. of Registration, State House, Springfield.

INDIANA: *Examination.* Indianapolis, June 17-19. Exec. Sec., Board of Medical Registration and Examination, Miss Ruth V. Kirk, 416 K. of P. Bldg., Indianapolis.

KANSAS: *Endorsement.* Kansas City, June 26. Sec., Board of Medical Registration and Examination, Dr. J. F. Hassig, 905 N. Seventh St., Kansas City 10.

KENTUCKY: *Examination.* Louisville, Sept. 22-24. Sec., State Board of Health, Dr. P. E. Blackerby, 620 S. Third St., Louisville 2.

MAINE: Augusta, July 1-2. Sec., Board of Registration in Medicine, Dr. Adam P. Leighton, 192 State St., Portland.

MARYLAND: *Medical Examination.* Baltimore, June 17-20. Sec., Dr. E. H. Kloman, 1215 Cathedral St., Baltimore. *Homeopathic.* Baltimore, June 10-11. Sec., Dr. J. A. Evans, 612 W. 40th St., Baltimore.

MASSACHUSETTS: *Examination.* Boston, July 8-11. Sec., Board of Registration in Medicine, Dr. H. Q. Gallupe, 413 E State House, Boston.

MICHIGAN: * *Examination.* Detroit, June 9-11. Semi-Annual meeting, Lansing, June 24. Sec., Board of Registration in Medicine, Dr. J. E. McIntyre, 100 W. Allegan St., Lansing 8.

MINNESOTA: * *Examination.* Minneapolis, June 17-19. Sec., Dr. J. F. DuBois, 230 Lowry Medical Arts Bldg., St. Paul 2.

MISSISSIPPI: Jackson, June 23-24. Asst. Sec., State Board of Health, Dr. R. N. Whitfield, Jackson 113.

MISSOURI: *Examination.* St. Louis, June 9-11. Exec. Sec., Mr. John A. Hailey, Box 4, State Capitol Building, Jefferson City.

MONTANA: Helena, Oct. 6-8. Sec., Dr. Otto G. Klein, 302 N. Main St., Helena.

NEBRASKA: * *Examination.* Omaha, June. Dir., Bureau of Examining Boards, Mr. Oscar F. Humble, State Capitol Bldg., Lincoln.

NEW HAMPSHIRE: *Examination.* Concord, Sept. 11-12. Sec., Board of Registration in Medicine, Dr. John S. Wheeler, 107 State House, Concord.

NEW JERSEY: *Examination.* Trenton, June 17-18. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW YORK: *Examination.* Albany, Buffalo, New York and Syracuse, June 23-26. Sec., Dr. Jacob L. Lochner Jr., State Education Bldg., Albany.

NORTH CAROLINA: *Endorsement.* Raleigh, June 24. *Examination.* Raleigh, June 23-26. Acting Sec., Mrs. L. McNeill, 226 Hillsboro St., Raleigh.

NORTH DAKOTA: Grand Forks, July 1-4. Sec., Dr. G. M. Williamson, 4½ S. Third St., Grand Forks.

OHIO: *Examination.* Columbus, June 17-20. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: * Oklahoma City, June 3-4. Sec., Dr. Frank Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

PENNSYLVANIA: *Examination.* Philadelphia and Pittsburgh, July 8-11. Final date for filing application is June 10. Acting Sec., Bureau of Professional Licensing, Department of Public Instruction, Mrs. M. G. Steiner, 351 Education Bldg., Harrisburg.

RHODE ISLAND: * *Examination.* Providence, June 19-20. Chief, Division of Professional Regulation, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH CAROLINA: Columbia, June 23-25. Sec., Dr. N. B. Heyward, 1329 Blanding St., Columbia.

SOUTH DAKOTA: * Pierre, July 15-16. Sec., Medical Licensure, State Board of Health, Dr. Gilbert Cottam, Capitol Bldg., Pierre.

TENNESSEE: * Memphis and Nashville, June 11-12. Sec., Dr. Harley W. Qualls, 1635 Exchange Bldg., 130 Madison Ave., Memphis 3.

TEXAS: Austin, June 5-7. Sec., Dr. T. J. Crowe, 918-20 Texas Bank Building, Dallas 2.

UTAH: Examinations are held in June of every year. Asst. Dir., Department of Registration, Miss Rena B. Loomis, 324 State Capitol Bldg., Salt Lake City 1.

VIRGINIA: Richmond, June 18-21. Sec., Dr. K. D. Graves, 306 Medical Arts Bldg., 30½ Franklin Rd., Roanoke.

WEST VIRGINIA: *Examination.* Charleston, June 30, July 1-2. Commissioner, Public Health Council, Dr. Newman H. Dyer, State Capitol, Charleston 5.

WYOMING: *Examination.* Cheyenne, June 2. Sec., Dr. G. M. Anderson, Capitol Bldg., Cheyenne.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ARIZONA: June 17. Sec., Mr. Francis A. Roy, Science Hall, University of Arizona, Tucson.

CONNECTICUT: June 14. Exec. Asst., State Board of Healing Arts, Mr. W. G. Reynolds, 250 Church St., New Haven 10.

DISTRICT OF COLUMBIA: *Examination.* Washington, Oct. 20-21. Sec., Commission on Licensure, Dr. G. C. Ruhland, 6012 E. Municipal Bldg., Washington.

IOWA: Des Moines, July 8. Sec., Dr. Ben H. Peterson, Coe College, Cedar Rapids.

MINNESOTA: Minneapolis, June 3-4. Sec., Dr. Raymond N. Bieter, 105 Millard Hall, University of Minnesota, Minneapolis 14.

SOUTH DAKOTA: Vermillion, June 6-7. Sec., Dr. G. M. Evans, 310 E. 15th St., Yankton.

TENNESSEE: *Examination.* Nashville and Memphis, June 13-14. Sec., Dr. O. W. Hyman, 874 Union Ave., Memphis.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Dentists: Extraction of Sixteen Teeth Not Necessarily Negligence.—The plaintiff sued for damages for the death of her husband allegedly caused by the negligence of the defendant dentist. From a judgment of no cause of action, the plaintiff appealed to the Supreme Court of Michigan.

The plaintiff's husband went to the office of the defendant dentist for the purpose of having sixteen upper teeth extracted. Twenty minutes later he was dead, after having been administered gas as an anesthetic. At the trial the defendant was not asked, nor did he state, how many teeth were actually extracted or how the death occurred. A physician testified that he had examined the deceased's heart not long before and found it normal. In this physician's opinion, the greater the number of teeth extracted, the greater the shock to the system, but in answer to a hypothetical question he said that the death could have resulted from any one of a number of causes.

Considering the evidence in the light most favorable to the plaintiff, can it be said that the deceased's death resulted from the surgical shock following the extraction of sixteen teeth or from the improper administration of gas, asked the court? In a prior Michigan case (*Voss* v. *Adams,* 271 Mich. 203, 259 N. W. 889) there was testimony to the effect that, according to local standards, it was not proper practice to extract more than eight teeth at one sitting because of the increased danger of surgical shock and the increased infection let loose in the system. In the instant case, said the court, there was no testimony in the record as to the standard practice in the locality and no showing of the cause of death. The mere proof that sixteen teeth had been extracted at one sitting, in the absence of testimony showing that this was not the usual and ordinary practice of dentists in the locality, is not, in and of itself, sufficient to establish negligence, added the court. Plaintiff had the burden of showing that her husband's death was brought about by the defendant's failure to exercise the reasonable and ordinary care, skill and diligence possessed by others in the same line of practice and work in similar localities. We are compelled, concluded the Supreme Court, to agree with the trial judge that the plaintiff failed to establish negligence on the part of the defendant. Accordingly the judgment in favor of the defendant was affirmed.—*Nemer v. Green, 25 N. W. (2d) 207 (Mich., 1946).*

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1937 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied with stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Heart Journal, St. Louis

33:1-134 (Jan.) 1947

Electrocardiographic Studies in Rheumatic Heart Disease with Reference to Interpretation of Multiple Unipolar Precordial Leads. S. R. First, A. W. Stickle and R. H. Bayley.—p. 1.
Duration of Electrical Systole (QT) in Acute Rheumatic Carditis in Children. L. M. Taran and Nelly Szilagyi.—p. 14.
Electrocardiographic Changes Associated with Methyl Alcohol Poisoning. A. S. Weisberger and J. A. MacLaughlin.—p. 27.
Atrionodal Rhythm with Ventricular Bigeminy: Report of Case with Unusual Mechanism. J. S. Perelman and R. Miller.—p. 34.
New Sensitive Portable Plethysmograph. G. E. Burch.—p. 48.
*Fatal Coronary Artery Disease in Young Men. W. D. Poe.—p. 76.
Effect of Sulfonamide Administration on Cardiac Function in Dog. Roberta Hafkesbring and Grace E. Wertenberger.—p. 84.
*Effect on Heart of Overdose of Epinephrine: Report of Case. A. P. McGinty and L. S. Baer.—p. 102.
*Case of Large Overdose of Epinephrine. O. Horwitz.—p. 107.
*Congenital Complete Heart Block Diagnosed in Utero with Sound Tracings and Simultaneous Electrocardiograph of the Mother. F. C. Jordan and H. Randolph.—p. 109.
Arteriosclerotic Aneurysm of Cardiac Coronary Arteries: Report of Case. N. Mitchell.—p. 112.
Congestive Heart Failure and Death in Case of Paroxysmal Auricular Tachycardia: Case Report. R. P. Grant.—p. 121.

Fatal Coronary Artery Disease in Young Men.—Coronary artery disease is not generally believed to be a frequent cause of death below the age of 40. Nine instances reported by Poe from the records of one army hospital comprised 7.4 per cent of all necropsies, a total of 121, performed on men between the ages of 18 and 40 years who died of natural causes. Few of these 9 young men gave evidence of having had symptoms of coronary disease before the fatal episode. Death was sudden and unheralded. Most of the 9 young men were of average or below average height. The weight of the heart did not reflect the seriousness of the cardiac condition. Slight to moderate enlargement of the liver may have been related to the heart disease. Evidence of old myocardial injury was frequent. Evidence of fresh infarction is rare in young men dying of coronary artery disease.

Effect of Overdose of Epinephrine.—McGinty and Baer report observations on a naval aviator who was inadvertently given an intramuscular injection of 5 cc. of a 1 : 1,000 solution of epinephrine hydrochloride. The arrhythmia noted in the first thirty minutes could have been due to frequent extrasystoles or to transient ventricular fibrillation. The precordial distress, the transient elevation of temperature, the increase of sedimentation rate and the prolonged electrocardiographic changes could have been due to actual infarction of the myocardium. The evidence in this case indicates that epinephrine in large doses causes relative coronary insufficiency in the normal adult.

Large Overdose of Epinephrine.—Horwitz observed a patient with urticaria who was given by mistake an intramuscular injection of 7 cc. of a 1 : 1,000 solution of epinephrine. There were no alarming immediate or late effects. There was no excessive perspiration and no cardiac arrhythmia other than a mild tachycardia. The highest blood pressure recorded was 220/96. The pulse rate reached 128 per minute. The epinephrine must have caused considerable angiospasm at the site of injection, thereby allowing itself to be absorbed slowly by the systemic circulation.

Congenital Heart Block Diagnosed in Utero.—Jordan and Randolph report that, because the fetal heart sounds five weeks before term were extremely slow, it was thought that the life of the fetus was in danger; but after observation for several hours with no change in the heart rate no attempt was made to induce labor. During the subsequent ten days the fetal heart rate was always between 40 and 46 per minute. Sound tracings of the fetal heart were made simultaneously with the maternal electrocardiogram. The mother was delivered at the beginning of the ninth month. The baby was an apparently healthy infant weighing 5 pounds 12 ounces (2,608 Gm.). Since birth the infant has presented a difficult feeding problem. Regurgitation of food is still frequent, yet fairly normal growth has taken place.

American Practitioner, Philadelphia

1:285-342 (Feb.) 1947

Diagnosis and Management of Acute Arthritis. W. D. Robinson.—p. 285.
Myocarditis in Infectious Diseases. I. Gore.—p. 292.
Present Status of Electroshock Therapy. M. Kleinerman.—p. 299.
Clinical Analysis of 550 Cases of Bacterial Meningitis: Diagnostic Features and Various Methods of Treatment. P. S. Rhoads.—p. 305.
Gastrointestinal Allergy. R. Chobot.—p. 315.
Renal Complications in Children Receiving Sulfonamide Drugs. H. B. Hutt.—p. 317.

American Review of Tuberculosis, New York

54:465-620 (Dec.) 1946

Heart Disease in Case Finding Program. H. F. West.—p. 465.
Cancer and Pulmonary Tuberculosis: Diagnostic Problems in Patients with Cancer of Lung in Presence of Pulmonary Tuberculosis. B. Gerstl, F. C. Warring Jr. and K. S. Howlett Jr.—p. 470.
*Cytology of Bronchial Secretions: Diagnostic Aid in Diagnosis of Pulmonary Tuberculosis. P. A. Herbut and L. H. Clerf.—p. 488.
Pneumothorax Fluid: Its Management by Systematic Aspiration. K. S. Howlett Jr. and H. L. Ehrenkrantz.—p. 495.
Cholesterol Pleural Effusion. L. C. Evander.—p. 504.
Extrapleural Pneumothorax in Silicotuberculosis. H. M. Maier and A. Hurst.—p. 509.
Tuberculosis—A Labor Problem. L. Price.—p. 512.
Mass Miniature Radiography: Survey in United States Army Air Forces. E. Wayburn.—p. 527.
Serial Tuberculin Tests and Stability of Tuberculin Reaction. Ruth R. Puffer, H. C. Stewart, R. S. Gass and E. F. Harrison.—p. 541.
*Certain Effects of Streptomycin in Mycobacteria in Vitro. G. Middlebrook and D. Yegian.—p. 553.
Synthesis of B Complex Vitamins by Tubercle Bacilli When Grown on Synthetic Media. Hilda Pope and D. T. Smith.—p. 559.
Relationship of Acquired Resistance, Allergy, Antibodies and Tissue Reactivities to Components of Tubercle Bacillus. S. Raffel.—p. 564.
Respiratory Patterns in Pulmonary Tuberculosis. M. N. Rao and L. Silverman.—p. 574.

Cytology of Bronchial Secretions in Pulmonary Tuberculosis.—Herbut and Clerf state that a presumptive diagnosis of pulmonary tuberculosis can be made from a cytologic study of smears made of bronchoscopically removed secretions and stained by the Papanicolaou technic. Rounded clusters of ciliated epithelial cells, single ballooned and vacuolated ciliated epithelial cells and giant cells of the Langhans type are of importance in the diagnosis of tuberculosis. The author describes the technic employed in preparing the smears and presents cases in which this procedure proved helpful. The method is of particular value in differentiating pulmonary tuberculosis from carcinoma in patients in whom the lesions are atypically situated, in whom there is little constitutional disturbance and in whom repeated examination of sputum fails to reveal tubercle bacilli.

Effects of Streptomycin on Mycobacteria in Vitro.—Middlebrook and Yegian describe the composition and preparation of the "Tween 80" medium, which is a liquid synthetic medium in which mycobacteria grow more rapidly and more diffusely than in the conventional mediums. Freshly isolated tubercle bacilli from patients with pulmonary tuberculosis appear to have a uniform susceptibility to streptomycin in vitro. Exposure of tubercle bacilli to streptomycin in relatively low concentration in vitro inhibits subsequent multiplication of multiplying but not of nonmultiplying tubercle bacilli when they are transferred to medium free of streptomycin. All mycobacteria thus far tested develop resistance to streptomycin in vitro at a rapid rate. Resistance to streptomycin persists for at least four months and appears not to be paralleled by diminution in virulence for guinea pigs. The acquisition of resistance of mycobacteria to streptomycin is independent of their acquisition of resistance to a sulfonamide (or a sulfonamide-like agent), and the development of resistance to these drugs is retarded when mycobacteria are exposed to the two types of agents simultaneously.

Annals of Internal Medicine, Lancaster, Pa.

26:191-340 (Feb.) 1947

Medicine in European Theater of Operations. W. S. Middleton.—p. 191.
Scrub Typhus in Dutch New Guinea. E. N. Irons and H. E. Armstrong.—p. 201.
*Treatment of Acute Bacterial Endocarditis with Penicillin. Freda Wilhelm, H. L. Hirsh, H. H. Hussey and H. F. Dowling.—p. 221.
*Cardiac Arrhythmias Complicating Total Pneumonectomy. E. Massie and A. R. Valle.—p. 231.
Rheumatoid Spondylitis: Study of 1,035 Cases. H. F. Polley and C. H. Slocumb.—p. 240.
*Trichinosis: Report of Epidemic. F. H. Hathaway and L. Blaney.—p. 250.
*Pleuropulmonary Tularemia: Observations on 12 Cases Treated with Streptomycin. J. S. Hunt.—p. 263.

Penicillin in Acute Bacterial Endocarditis.—Eight patients who had acute bacterial endocarditis were treated by Wilhelm and her associates with penicillin. Five patients died, the diagnosis being confirmed by necropsy. The clinical course of the 3 patients who recovered satisfied the criteria established for the diagnosis of this disease. The 3 patients who recovered had staphylococcic endocarditis, while of the 5 who died 4 had staphylococcic and 1 had pneumococcic endocarditis. The only hope in this disease lies in early diagnosis and vigorous treatment. Of the 5 patients who died, 4 probably were inadequately treated. The diagnosis was established late in the course of the disease in 3 of these patients, so that penicillin had been administered for two days or less before death. Whenever possible, the sensitivity of the causative organism should be determined and the dose of penicillin established accordingly. If the sensitivity of the organism to penicillin has not been determined, doses of 500,000 units per day should be given.

Cardiac Arrhythmias Complicating Pneumonectomy.—In an analysis of 120 patients undergoing total lung resection, Massie and Valle found that 11 developed arrhythmia of sufficient severity to have endangered the outcome of the operation or at least to have caused symptomatic difficulty. The arrhythmias included 5 instances of auricular fibrillation, 4 of auricular flutter and 1 each of frequent auricular and ventricular extrasystoles. The reason for the high incidence of arrhythmias following pneumonectomy could not be definitely determined. It is suggested that anoxemia and vagal stimulation may act synergistically in the production of these abnormal cardiac rhythms. Constant observation must be maintained for early recognition of the occurrence of abnormal cardiac rhythms. Treatment should be directed primarily to the attainment of prompt restoration of a normal sinus rhythm, and this result may be most often accomplished by rapid intravenous digitalization.

Epidemic of Trichinosis.—Hathaway and Blaney studied an epidemic of trichinosis among 587 German prisoners of war. Eighty-three were observed in the hospital; 437 who were not hospitalized had blood smears taken approximately one week after the last improperly prepared meal. The presence of eosinophilia in a high percentage of the men not hospitalized suggested the presence of trichinosis in a mild or subclinical form in 100 men. The exact date the trichinous meat was served cannot be determined, but sausage had been generally served uncooked. The typical clinical picture was characterized by a sudden onset of chilliness, fever, malaise and frontal headache. Associated with these complaints or occurring shortly afterward there was puffiness of the eyelids, which generally lasted three to five days. In most instances muscle pain, or muscle pain with tenderness, occurred a few days later but had disappeared or diminished considerably at the time of discharge. The average hospital stay was twenty-six days. The most significant laboratory finding was eosinophilia, which ranged as high as 61 per cent. The fact that this epidemic resulted after the ingestion of meat from reliable packers indicates that all pork products, regardless of the source, are potentially dangerous from the standpoint of trichinosis if served improperly cooked.

Pleuropulmonary Tularemia.—Hunt says that, during the period of the study of streptomycin therapy conducted in Nashville hospitals from April to September 1946, 15 cases of pleuropulmonary tularemia were encountered. Twelve of these patients were treated with streptomycin. The patients showed a prompt clinical response to small doses of the antibiotic. The rapid disappearance of Pasteurella tularensis from the sputum or pleural fluid, as determined by animal inoculation, indicated that the organism was rapidly killed in the tissues. The regression of pulmonic lesions and the healing of primary ulcers were believed to have been accelerated by streptomycin treatment but required a longer time interval than did clinical recovery. All the patients presented a rise in serum agglutinin titer after treatment. This increase in the serum agglutinin level may have resulted from the antigenic stimulus provided by the destruction of large numbers of organisms. Because of the diagnostic difficulties presented by many cases of tularemic pneumonia and the prompt response of tularemia to streptomycin therapy, the value of a therapeutic trial with this antibiotic in severe pneumonia of undetermined origin in areas in which tularemia is endemic is apparent.

Journal of Allergy, St. Louis

17:329-418 (Nov.) 1946

Studies in Food Allergy: V. Antigenic Relationship Among Members of Fish Family. L. Tuft and G. I. Blumstein, with technical assistance of V. M. Heck.—p. 329.
Measurement of Circulating Ragweed Antibodies and Antigen: Correlation with Symptoms and Treatment. H. L. Alexander, M. C. Johnson and J. H. Alexander.—p. 340.
Treatment of Residual Lymphoid Tissue in Nasopharynx by Radium. L. N. Gay.—p. 348.
Treatment of Intractable Asthma. A. L. Barach.—p. 352.
General Adaptation Syndrome and Diseases of Adaptation. H. Selye.—p. 358.

Journal of Immunology, Baltimore

55:103-204 (Feb.) 1947

Complement Fixation Test in Yellow Fever Epidemiology: Use of Globulin Antigen in Immunity Surveys. A. Perlowagora and T. P. Hughes.—p. 103.
Further Experimental Studies on Role of Plasma Cells as Antibody Producers. M. Bjørneboe, H. Gormsen and F. Lundquist.—p. 121.
Specific Antigen Recovered from Tissue Infected with M. Paratuberculosis (Johne's Bacillus): III. Further Studies on Nature of Antigen and on Methods for Demasking It. B. Sigurdsson.—p. 131.
Transition Forms of Blood Groups. L. Hirszfeld.—p. 141.
Antigenic Specificity of Syphilitic Antibody Globulin. S. D. Henriksen.—p. 153.
Relationship of Sulfhydryl and Disulfide Groups to Antigenic Power of Bovine Serum Albumin. R. R. Williams.—p. 161.
Complement and Opsonic Activities of Fresh Human Serums. E. E. Ecker and G. Lopez-Castro.—p. 169.
Detoxification of Crystalline Botulinum Type A Toxin. G. A. Hottle and A. Abrams.—p. 183.
Further Study of Quantitative Complement Fixation Test as Applied to Serum Diagnosis of Typhus Fever. Florence M. Varley and F. R. Weedon.—p. 189.
Specific Complement Fixing Diagnostic Antigens for Neurotropic Viral Diseases. C. J. DeBoer and H. R. Cox.—p. 193.

Journal of Infectious Diseases, Chicago

80:1-120 (Jan.-Feb.) 1947

Studies on Infection with Bacillus Anthracis: I. Histopathologic Study of Skin Lesions Produced by B. Anthracis in Susceptible and Resistant Animal Species. W. J. Cromartie, W. L. Bloom and D. W. Watson.—p. 1.
Id.: II. Immunologic and Tissue Damaging Properties of Extracts Prepared from Lesions of B. Anthracis Infection. W. J. Cromartie, D. W. Watson, W. L. Bloom and R. J. Heckly.—p. 14.
Id.: III. Chemical and Immunologic Properties of Protective Antigen in Crude Extracts of Skin Lesions of B. Anthracis. D. W. Watson, W. J. Cromartie, W. L. Bloom, G. Kegeles and R. J. Heckly.—p. 28.
Id.: IV. Preparation and Characterization of Anthracidal Substance from Various Animal Tissues. W. L. Bloom, D. W. Watson, W. J. Cromartie and M. Freed.—p. 41.
Studies on Bovine and Human Leptospirosis. H. Bernkopf, L. Olitzki and L. A. Stuczynski.—p. 53.
Laboratory Infection with Psittacosis Virus Treated with Penicillin and Sulfadiazine, and Experimental Data Bearing on Mode of Infection. T. Rosebury, H. V. Ellingson and G. Meiklejohn, with assistance of F. Schabel.—p. 64.
Asexual Reproduction of Plasmodium Cynomolgi in Rhesus Monkeys. W. H. Taliaferro and Lucy Graves Taliaferro.—p. 78.
Study of Aerobic Streptococci Isolated from Uterus and Vagina. K. Eileen Hite and H. C. Hesseltine.—p. 105.
*Typhoid Fever in Vaccinated Laboratory Workers. T. A. Haedicke.—p. 113.
Further Evaluation of Skin Test for Filariasis in Man, Based on Results Obtained in British Guiana. D. R. A. Wharton.—p. 117.

Typhoid in Laboratory Workers.—Of the 4 patients studied by Haedicke 2 acquired the disease while working in the typhoid vaccine laboratory at the Army Medical School. These 2 patients worked with material containing 35 billion typhoid

bacilli per cubic centimeter. The other 2 patients acquired their infection in the typhoid research division of the Army Medical School. There was no accident to which these infections could be attributed. These patients had all received at least one complete series of typhoid vaccine prior to the onset of their illness. No relationship was found to exist between the date of the last immunization and the date of onset of the disease. The interval varied from twenty-eight days to eleven months. The diagnosis was difficult in all cases because of the absence of the usual signs and symptoms. The clinical course was less severe than that usually seen in persons who have not been vaccinated. All patients were only mildly ill. The average length of time during which the temperature was elevated was five and a half weeks. There were no complications.

Journal-Lancet, Minneapolis

47:47-82 (Feb.) 1947

Progress in Antibiotic Therapy. C. S. Keefer.—p. 50.
Progress in Allergy Therapy. S. M. Feinberg.—p. 52.
Progress in Fungous Disease Therapy. L. Schwartz.—p. 56.
Progress in Rickettsial Disease Therapy, with Special Reference to Para-Aminobenzoic Acid. A. Yeomans.—p. 60.
General Medical Aspects of Atomic Energy. F. H. Krusen.—p. 64.
Nuclear Physics and Medical Research. G. Failla.—p. 68.
Physiologic Reaction to Radioactive Isotopes. S. Warren.—p. 69.

47:83-124 (March) 1947

Common Dermatologic Diseases. M. G. Fredericks.—p. 83.
Orientation in Gynecologic Uterine Bleeding. C. E. McLennan.—p. 90.
Roentgenographic Aids in Carpal Scaphoid Fractures. R. E. Van Demark.—p. 94.
Few Essentials in Prescribing Physical Medicine in General Practice. E. C. Elkins.—p. 97.
Psychosomatic Diagnosis. G. R. Kamman.—p. 102.

Journal of Thoracic Surgery, St. Louis

16:1-98 (Feb.) 1947

Surgical Thoracic Tumors in Navy Personnel. W. L. Watson and H. D. Diamond.—p. 1.
Chest Tumor Registry. J. E. Ash.—p. 12.
Report of 196 Lobectomies Performed at Kennedy General Hospital Chest Surgical Center from 1943 to 1946 with One Death. R. H. Meade Jr., E. B. Kay and F. A. Hughes.—p. 16.
*Follow-Up of Cardiopulmonary Function in Four Young Individuals After Pneumonectomy. A. Cournand, A. Himmelstein, R. L. Riley and C. W. Lester.—p. 30.
Use of Curare in Anesthesia for Thoracic Surgery. H. B. Stephens, P. Harroun and F. E. Beckert.—p. 50.
*Surgical Treatment of Myasthenia Gravis. O. Theron Clagett and L. M. Eaton.—p. 62.
Aerosol Penicillin as Therapeutic Adjunct in Preparation of Patients with Suppuration of Lung for Pulmonary Resection. C. W. Findlay Jr. and R. H. Sweet.—p. 81.
Anesthesia for Thoracic Surgery in United States Army. L. H. Mousel.—p. 91.

Cardiopulmonary Function After Pneumonectomy.—Cournand and his co-workers followed through the period of growth the cardiopulmonary function of 4 patients who had undergone pneumonectomy during childhood or early adolescence. They studied at regular intervals the lung volumes, the ventilatory efficiency of the chest bellows, the efficiency of alveolar ventilation and the state of respiratory gas exchange in the lung and in the arterial blood at rest and during exercise. The right ventricular pressures and the pulmonary blood flow were measured in 3 of the 4 subjects. In 2 patients who developed moderate pulmonary distention the only significant observations were a reduction in breathing reserve and severe oxygen unsaturation in the arterial blood during strenuous exercise. The moderate degree of pulmonary distention, which increased slightly in 1 subject during growth of the lung, has not progressed during the last three years. One subject without pulmonary distention had the same performance as a normal person. No significant hypertension has developed in the lesser circulation.

Surgical Treatment of Myasthenia Gravis.—This report by Clagett and Eaton of the Mayo Clinic is based on a series of 32 patients with myasthenia gravis who have been followed for six or more months since operation on the thymus gland. The entire series consisted of 191 patients, 159 of whom were not operated on. In 15 of the 32 cases thymic tumors were found at operation. In all but 1 of the remaining 17 a good sized thymus gland was found and removed. The authors believe that the results to date warrant thymic exploration in all cases of myasthenia gravis in which the disease is severe or progressive and in which the condition will permit surgery. The sternum splitting approach offers the best exposure in most instances.

Kentucky Medical Journal, Bowling Green

45:1-30 (Jan.) 1947

Medical Management of Hypertension. W. B. Troutman.—p. 7.
Fundus Changes in Hypertension. H. A. Pfingst.—p. 9.
Surgical Treatment of Hypertension. E. G. Grantham.—p. 10.
Streptomycin in Treatment of Urinary Tract Infections. W. V. Pierce.—p. 17.
Evaluation of Present Day Treatment of Cancer of Stomach. C. C. Johnston.—p. 21.

45:31-56 (Feb.) 1947

Pathogenesis and Treatment of Essential Hypertension. R. C. Blount.—p. 34.
What Does Prostigmine Offer to Victims of Cerebral Palsy? T. M. Marks.—p. 40.

Symposium on Fluid Balance as a Pediatric Problem

Effect of Diarrhea on Water Balance in Infants and Children. J. W. Bruce.—p. 43.
Effect of Vomiting on Water Balance in Infants and Children. W. S. Ogden.—p. 45.
Clinical Physiology of Water Balance in Infants and Children. H. Lawson.—p. 47.

Medicine, Baltimore

26:1-114 (Feb.) 1947

Tropical Deterioration and Nutrition: Clinical and Biochemical Observations on Troops. R. M. Kark, H. F. Aiton, E. D. Pease and others.—p. 1.
Neurologic Conditions Resulting from Prolonged and Severe Dietary Restriction: Case Reports in Prisoners of War, and General Review. D. Denny-Brown.—p. 41.

Minnesota Medicine, St. Paul

30:121-224 (Feb.) 1947

Use of Insulin Mixtures. R. G. Sprague and L. O. Underdahl.—p. 153.
Present Southern Minnesota Medical Association: Its Antecedents, Purposes and Character. C. B. McKaig.—p. 157.
*Glomus Tumors: Report of 2 Cases. H. W. Meyerding and J. H. Varney.—p. 159.
Chronic Mastoiditis with Cholesteatoma and Stenosis of External Auditory Meatus: Report of 2 Cases. H. I. Lillie and J. B. McBean.—p. 161.
Further Observations on Prognosis in Angina Pectoris Due to Coronary Sclerosis: Study of 405 Patients Who Survived Ten or More Years. G. E. Montgomery Jr., T. J. Dry and R. P. Gage.—p. 162.
Polyneuritis: Differentiation of Infectious Polyneuritis (Guillain-Barré Syndrome) and the Neuritis of Porphyria. C. L. Yeager.—p. 166.
Industrial Integration. A. E. Ritt.—p. 174.

Glomus Tumors.—Meyerding and Varney point out that glomus enlargements, or arteriovenous shunts, occur normally in skin and subcutaneous tissue. They occur most frequently in the finger tips. With increased growth of these tumors they become excruciatingly painful. They are usually located under the nail. Because of their rarity they may remain unrecognized for years. The authors have operated on 2 patients who had glomus tumors of nine and sixteen years' duration respectively. In each case there was a complete relief of symptoms. Early diagnosis and eradication are dependent on the recognition of the lesion by the general practitioner.

Missouri State Medical Assn. Journal, St. Louis

44:163-240 (March) 1947

*Dissolution of Staghorn Calculus in Kidney Pelvis by Intermittent Irrigation with Solution G. J. J. Cordonnier.—p. 179.
Ear Canal Infections. O. J. Dixon.—p. 181.

Dissolution of Staghorn Calculus with Solution G.—Cordonnier says that solution G is a combination of citric acid, magnesium oxide and sodium carbonate in water. In view of the technical difficulty of a second operation on the left side of a woman aged 26 and the relatively poor condition of the right kidney, it was felt that dissolution of the left renal calculus should be attempted. Two number 6 F. catheters were passed to the kidney pelvis and left in place. Continuous irrigation with solution G was instituted. At the end of thirty-six hours the patient developed severe renal colic, gross hematuria and fever, and irrigations were discontinued. She then was placed on an acidification regimen using ammonium chloride, for two

months. After this period the stone appeared less dense. The same procedure was followed as before, and irrigations with solution G were maintained for one hundred and twenty hours, when pain, fever and gross hematuria again prevented further treatment. At this time a great deal of the stone was found to have disappeared. She was again discharged on an acidification regimen and returned after one month, at which time the shadow in the pelvis was found to have disappeared.

New England Journal of Medicine, Boston

236:305-340 (Feb. 27) 1947

The Physician and School Medical Services. G. M. Wheatley.—p. 305.
Physical Rehabilitation of Disabled Persons. G. G. Deaver.—p. 311.
Comparative Study of Penicillin X and G and Crystalline Penicillin G. Georgine Rotman-Kavka, H. L. Hirsh and H. F. Dowling.—p. 314.
Treatment of Chronic Osteomyelitis with Streptomycin: Preliminary Report. T. A. Lamphier and C. Cashman.—p. 318.
Psychiatry. V. P. Williams.—p. 322.
Dissecting Aneurysm of Aorta, with Rupture into Left Pleural Cavity and with Localized Expansion and Partial Healing.—p. 327.
Periosteal Fibrosarcoma of Jaw.—p. 330.

Northwest Medicine, Seattle

46:89-168 (Feb.) 1947

Treatment of Recurrent Peptic Ulcers Following Gastric Operations. M. E. Steinberg.—p. 114.
Advanced Carcinoma of Stomach Requiring Combined Gastrectomy and Colectomy. T. W. Buschmann.—p. 126.
Use of Hormones in Gynecologic and Obstetric Practice. E. S. Taylor.—p. 128.
Evaluation of Present Day Methods and Treatment of the Mentally Ill. N. K. Rickles.—p. 134.

46:169-248 (March) 1947

Infectious Mononucleosis: Some Aspects of Its Etiology. W. M. Brock.—p. 197.
Malignant Melanoma with Generalized Skin Blackening: The White Girl Who Turned Black. D. V. Truehlood.—p. 199.
Modern Surgical Methods. T. L. Hyde, W. N. Morse, L. V. Moore and J. H. Skirving.—p. 202.
Postoperative Parenteral Fluids, with Particular Reference to Use of Saline and Amino Acids. J. A. Aalpoel.—p. 205.
Drug Toxicity in Renal and Hepatic Disease. D. M. Green.—p. 210.
High External Gastrostomy Opening in Upper Esophageal Obstruction. J. K. Poppe.—p. 211.
Ringworm of Scalp in Pacific Northwest. L. F. Ray.—p. 214.
*Chromaffin Tissue Tumors and Associated Paroxysmal Hypertension. H. O. Schneider.—p. 216.

Chromaffin Tissue Tumors and Paroxysmal Hypertension.—A man aged 41 complained of attacks of faintness, flushing, palpitation and a choking sensation whenever stooping over. The only abnormal finding was a firm, smooth mass in the right upper abdominal quadrant. The patient complained of his presenting symptoms while the mass was being palpated. He became pale, restless and apprehensive. The pulse rate increased from 72 to 144 per minute. Blood pressure was 210/130. All symptoms and abnormal findings shortly returned to normal after the abdominal examination was completed. These findings could be produced repeatedly by this manipulation. At operation a large tumor was found located in the region of the mesenteric ganglions. The tumor was removed. The pathologic diagnosis was benign paraganglioma. Since his discharge from the hospital the patient has remained symptom free and has no evidence of hypertension. Schneider reports this case of a palpable intra-abdominal chromaffin tissue tumor to focus attention on the possible existence of such tumors in the presence of hypertension.

Puerto Rico J. Pub. Health & Trop. Med., San Juan

22:139-232 (Dec.) 1946

*Experimental Chemotherapy of Filariasis Bancrofti. J. T. Culbertson, H. M. Rose, F. H. Morales, J. O. Gonzalez and Caroline Kreiss Pratt.—p. 139.
Quantitative Determination of Schistosoma Mansoni Ova in Feces from Patients Under Treatment with Antimonial Drugs. J. O. Gonzalez and F. H. Morales.—p. 210.
Treatment of Schistosomiasis Mansoni: Evaluation of Parasitotropic Effects of Fuadin and Tartar Emetic. F. H. Morales, Caroline Kreiss Pratt and J. O. Gonzalez.—p. 224.

Chemotherapy of Filariasis Bancrofti.—Culbertson and his associates summarize the results of a study designed to discover chemotherapeutic agents effective in filariasis bancrofti. Preliminary tests were conducted on a naturally occurring filariasis (Litomosoides carinii) in the cotton rat. One hundred and fourteen patients with filariasis bancrofti have been treated with various drugs, 35 being treated with neostibosan, 11 with neostam, 6 with urea stibamine, 5 with stibanose, 15 with fuadin, 20 with anthiomaline, 4 with antimony and potassium tartrate and 18 with melarsen oxide. Fifteen patients have been left untreated to serve as a control group. Filariasis bancrofti can be eliminated from patients by the administration of certain compounds of antimony or arsenic. The pentavalent antimonial neostibosan shows the greatest promise because it exerts a strong antagonistic effect on the filarial worm and can be used with comparative safety, being well tolerated by all subjects.

Public Health Reports, Washington, D. C.

62:293-324 (Feb. 28) 1947

Comparative Study of Live and Killed Vaccines in Experimental Tuberculosis. Preliminary Note. B. J. Olson, K. Habel and W. R. Piggott.—p. 293.
Control of Anopheline Mosquito Larvae by Use of DDT-Oil Mists. F. F. Ferguson, E. H. Arnold and W. M. Upholt.—p. 296.
Inactivation of DDT Use in Anopheline Mosquito Larvicides. W. M. Upholt.—p. 302.
*Isolation of Unidentified Spirochete From Hen's Eggs After Inoculation with Liver Tissue from Hens. E. A. Steinhaus and L. E. Hughes.—p. 309.

62:325-368 (March 7) 1947

Evolution of Official Tuberculosis Control in the United States. R. G. Paterson.—p. 336.

62:369-412 (March 14) 1947

Health Insurance Programs and Plans of Western Europe: Summary of Observations. J. W. Mountin and G. St. J. Perrott.—p. 369.

Isolation of Unidentified Spirochete From Hen's Eggs.—The observations reported by Steinhaus and Hughes were made at the Rocky Mountain Laboratory near Hamilton, Mont., incidental to checking for the occurrence of disease in local flocks from which eggs were being obtained for laboratory use. This is the first known evidence of the occurrence of a spirochete in the tissues of hens in the United States. It has been felt desirable to report this finding, since it is of potential interest to laboratory workers using chick embryos for culturing various pathogens or for manufacturing vaccines.

Radiology, Syracuse, N. Y.

48:1-106 (Jan.) 1947. **Partial Index**

*Treatment of Malignant Tumors of the Testes: Review of 100 Cases. G. M. Kelby and K. W. Stenstrom.—p. 1.
Simultaneous Radiographic and Gastroscopic Examination of Stomach. C. L. Ould and M. E. Dailey.—p. 8.
Malignant Melanoma in Infancy: Report of 3 Cases. P. B. Russo.—p. 15.
The "Last Straw" Factor in Low Back Disability: Summary of 100 Cases Examined and Evaluated by Medical Advisory Board of the Industrial Commission of Arizona, 1934-1943. W. W. Watkins.—p. 20.
Diffuse Calcification of Pancreas. A. J. Present and M. J. Geyman.—p. 29.
Obstructive Hydronephrosis Produced by Aberrant Blood Vessels and Diagnosed by Intravenous Urography. J. Abowitz.—p. 33.
Practical Technic for Visualization of Bronchial Tree. G. W. Heublein and C. D. N. Gilfillan.—p. 37.
Cephalopelvimetry. L. A. Scarpellino.—p. 45.

Treatment of Malignant Tumors of Testes.—Kelby and Stenstrom analyzed 100 cases of malignant tumors of the testes observed at the University of Minnesota Hospitals from 1926 to 1943 inclusive. The group consisted of 34 unclassified cases, 37 seminomas and 29 carcinomatous mixed tumors. Metastasis was known to be present before irradiation was started in 65 per cent of the seminomas, 86 per cent of the carcinomatous mixed tumors and 70 per cent of the unclassified group. Therapy consisted in simple orchiectomy and irradiation in 90 cases, irradiation alone in 6 advanced cases and Hinman's operation and irradiation in 4 cases. In seminomas of the testis the five year survival rate is 56 per cent. In the carcinomatous mixed tumor group only 1 patient survived five years, while in the unclassified group 29 per cent survived five years. A five year survival rate of 29 per cent was obtained in all the patients with malignant testicular tumors considered together.

Wisconsin Medical Journal, Madison

46:297-384 (March) 1947

New Methods for Control of Air Borne Infection, with Especial Reference to Use of Triethylene Glycol Vapor. O. H. Robertson.—p. 311.
Injuries Due to Cold, with Particular Reference to Frostbite and Trench Foot. H. B. Schumacker Jr.—p. 317.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

Archives of Disease in Childhood, London

21:121-180 (Sept.) 1946

Congenital Tuberculosis. Mary R. Hughesdon.—p. 121.
Complications of Localized Pulmonary Emphysema in Childhood. S. van Creveld and J. van den Broecke.—p. 139.
Etiology and Effects of Minor Grades of Anemia in Young Children: Studies at Group of Wartime Day Nurseries. Helen M. M. Mackay, R. H. Dobbs, K. Bingham and W. J. Martin.—p. 145.
Dermatomyositis in Childhood: Report of 4 Cases. N. S. Clark.—p. 160.
Relationship of Blood Pyruvic Acid Level to Deficiency States in Infancy and Childhood. E. C. Allibone and E. Finch.—p. 165.
Acute Bilateral Suprarenal Hemorrhage. G. C. Arneil.—p. 171.

British Journal of Tuberculosis, London

41:1-32 (Jan.) 1947

Technic and Standardization of Radiographs of Tuberculosis of Chest. J. F. Brailsford.—p. 1.
Bronchostenosis in Pulmonary Tuberculosis: Problem in Treatment. M. Davidson.—p. 7.
Treatment of "Tension Cavities." A. Bernou.—p. 11.
Pulmonary Tuberculosis: Effect of Sea Voyage on 37 Unselected Cases. R. Hambridge.—p. 15.
Selecting Treatment for Patient with Pulmonary Tuberculosis. J. L. Collis.—p. 20.

British Medical Journal, London

1:245-284 (Feb. 15) 1947

Water and Salt Depletion. H. L. Marriott.—p. 245.
Therapeutic Possibilities of Radiophosphorus. J. S. Mitchell.—p. 250.
*Pink Disease: Review of 65 Cases. T. N. Fisher.—p. 251.
*Pellagrous Encephalopathy. P. R. Graves.—p. 253.

Pink Disease.—Fisher reviews the histories of 65 patients who attended the outpatient clinic at the Royal Manchester Children's Hospital during the fourteen years from 1932 to 1945. The average duration in the infants recovering from pink disease was four months. The disease develops insidiously and is usually well established when the infant enters the hospital. There are three basic symptoms: a profound change in temperament, muscular hypotonia and tachycardia. Muscular hypotonia may be extreme. The art of walking, if already attained, is lost. The cutaneous symptoms include the vasomotor phenomena of the extremities, the rash, excessive perspiration and the special manifestations affecting the buccal and conjunctival mucous membranes. The puffy, pink or bluish red, cold, clammy hands and feet and the sudaminal eruption on the trunk and limbs, crisscrossed by excoriation from scratching, are the two most regularly appearing features. There were 9 deaths in this series. The precise cause of the disease is unknown and treatment has been symptomatic, with special emphasis on prevention of secondary infections. Home nursing is the method of choice. A high vitamin intake has been advised as a measure of protection against superimposed infection. The main principles in management are scrupulous attention to bodily hygiene, particularly when desquamation is in process, segregation from any human source of nasopharyngeal or respiratory infection, and patience in feeding. All food should be boiled or cooked and served as cold as possible.

Pellagrous Encephalopathy.—Graves describes a deficiency syndrome which was present among prisoners of war in Singapore. The clinical manifestations suggested a brain lesion: the only pathologic support for this contention was the naked-eye appearances of the brain and spinal cord at the necropsies in the fatal cases. No microscopic examination was possible. The syndrome occurred in the form of a comparatively small outbreak after large epidemics of vitamin B_1 deficiency resulting in neuritic and edematous beriberi and Wernicke's encephalopathy and later epidemics of B_2 complex deficiencies giving rise to angular stomatitis, glossitis, scrotal dermatitis, pellagra, dermal lesions, retrobulbar neuritis and the painful foot syndrome. The observations presented in this paper are based on 63 cases, of which 6 were fatal. When severe, this condition is responsible for widespread disorder of cerebral function resulting in disturbance of memory, coma with quadriplegic rigidity characteristic of a decorticate animal, and in some cases convulsions. As recovery occurred spastic paraplegia was the common residual state. In minor disorders less evidence of the initial cerebral illness may yet be followed by a crippling spastic paraplegia or a lesser degree of spastic weakness. Complete recovery may ensue after a severe initial onset.

Lancet, London

1:201-240 (Feb. 8) 1947

Prevention of Acute Respiratory Diseases, with Particular Reference to Influenza. C. H. Stuart-Harris.—p. 201.
Malnutrition in Indian Prisoners of War in Far East: Survey of 2,000 Cases. J. H. Walters, R. J. Rossiter and H. Lehmann.—p. 205.
Amyloid Macroglossia: Report of Case. Margaret D. Baber.—p. 210.
Production of Substance B by Corynebacterium Diphtheriae: Relation to Type of Strain and Severity of Toxemia. R. A. Q. O'Meara, R. S. W. Baker and H. H. Balch.—p. 212.
Bilateral Ureterocolostomy: Review of 17 Cases. A. C. Lovett-Campbell.—p. 214.
Outbreak of Weil's Disease. W. R. Gauld.—p. 216.

1:241-276 (Feb. 15) 1947

*Thyroidectomy and Thiouracil in Toxic Goiter: Interim Comparison. H. P. Himsworth, M. E. Morgans and W. R. Trotter.—p. 241.
Blood Volume Changes in Protein Deficiency. J. H. Walters, R. J. Rossiter and H. Lehmann.—p. 244.
Loiasis. R. D. C. Johnstone.—p. 250.
Painful Nodular Breast: Plea for Term Fibroadenosis. H. J. B. Atkins.—p. 253.
*Penicillin Prophylaxis in Acute Rheumatism. P. J. Burke.—p. 255.

Thyroidectomy and Thiouracil in Toxic Goiter.—Himsworth and his associates assess the relative values of thyroidectomy and thiouracil. Sixty-five cases of toxic goiter that were treated with the thiouracil series of drugs were followed for periods of up to thirty months and compared with 93 cases treated with subtotal thyroidectomy. The reduction in pulse rate and basal metabolic rate during the first three weeks of treatment with thiouracil was similar to that produced by thyroidectomy in the same period but was probably of lesser degree. From three to thirty months after the beginning of treatment there was no appreciable difference in the condition of the patients treated with thiouracil and those treated with subtotal thyroidectomy. Continued use of thiouracil drugs appears to be a good alternative treatment of toxic goiter. Thyroidectomy is advisable when tracheal obstruction is present, when a drug reaction occurs and when the patient is unable or unwilling to attend regularly for an indefinite period. After the administration of thiouracil is stopped, relapses occur. The longer patients were followed after the cessation of treatment, the more frequently did relapse occur. It is impossible as yet to say whether such remission can be permanent.

Penicillin Prophylaxis in Acute Rheumatism.—Burke used penicillin by mouth as a prophylactic against attacks of pharyngitis, particularly those due to streptococcic infections, in an effort to prevent the recurrence of acute rheumatic manifestations in susceptible patients. Twenty patients who had had acute rheumatism within the previous twelve months were divided into two groups of 10. The patients in the treated group were given three pastilles, each containing 500 Oxford units of calcium penicillin, to suck each day. They were instructed to place the pastilles between the cheek and the lower gum, to allow them to dissolve slowly, and to swallow the saliva containing the penicillin in solution. After the prophylactic treatment had been continued for a year it was evident that rheumatic manifestations were five times and throat infections six times more frequent in the control group than in the treated group.

Medical Journal of Australia, Sydney

1:193-224 (Feb. 15) 1947

*Basophilic Stippling of Red Corpuscles with Special Reference to Its Occurrence During Chrysotherapy. L. J. A. Parr and Eva A. Shipton.—p. 193.
Heterospecific Pregnancy: II. Incompatibility of ABO Groups. P. M. de Burgh, Ruth A. Sanger and R. J. Walsh.—p. 199.
Description of Modified Apparatus for Continuous Gastric or Duodenal Aspiration, with Brief Historical Review. P. W. Gill.—p. 201.
Note on Selection of Medical Students. E. S. Meyers and D. H. Meyers.—p. 203.

Basophilic Stippling, Especially During Chrysotherapy.—Parr and Shipton studied 20 patients taken at random from a series undergoing chrysotherapy. Basophilic stippling in the blood was revealed in all irrespective of the age or sex of the patient or of the variety of arthritis. The cases of rheumatoid

arthritis investigated were divisible into two main types, those in which sweating and clammy cyanotic hands were present and those in which the skin was dry and normal and no sweating or cyanosis was present. Many of the patients who had a dry skin and only slightly raised blood sedimentation rate proved to be ideal for chrysotherapy with regard to both the clinical response to treatment and the absence of toxicity. The authors suggest that patients showing severe anemia, eosinophilia, increased blood sedimentation rate, a shift of the neutrophil cells to the left and cold, clammy, cyanosed hands or dehydration should be carefully checked from time to time for stippled cells in their blood to prevent the onset of deleterious reactions.

Thorax, London

2:1-64 (March) 1947

Some Sequels of Abnormality at Thoracic Outlet. J. R. Learmonth. —p. 1.

Treatment of Pulmonary Hydatid Disease. N. R. Barrett.—p. 21.

Cavernous Haemangioma of Lung. W. Whitaker.—p. 58.

Progrès Médical, Paris

75:49-80 (Feb. 10) 1947

*Myeloma Following Bone Trauma and Osteomyelitis. M. Loeper, J. Vignalou and Mrs. T. Boreau.—p. 51.

Treatment of Chronic Bronchitis and of Pulmonary Emphysema with Aerosols on a Sodium Hyposulfite Base. L. Binet and F. Contamin. —p. 52.

Nosologic Considerations on Pain in Disturbances of Liver and of Biliary Tract. L. Cornil, H. Walther and G. F. Bonnet.—p. 54.

Myeloma Following Bone Trauma and Osteomyelitis.—Loeper and his co-workers report the occurrence of myeloma in a woman and in 2 men aged 52 and 30. The woman had sustained a fracture of the arm five years before sternal puncture revealed cells of definite plasmocytic type, and examination of the urine demonstrated the Bence Jones body and x-ray examination revealed multiple cavities of the humerus, the femur and the tibia. The older man sustained a fracture of the femur five years before plasmocytosis was revealed on sternal puncture. Urinalysis revealed Bence Jones albumosuria, but x-ray examination was negative. In the younger man the myeloma resulted from an osteomyelitis of twenty-five years' duration, with a residual fistula through which the bone tumor became exteriorized. In these 3 cases, in contrast to Bloodgood's observations, the trauma or the infection did not reveal a primary disease of the bone. The trauma and the chronic infection were instrumental in producing the myeloma, supporting Coley's observations and Kjurkoff's concept.

Cardiologia, Basel

11:111-174 (No. 3) 1946-47

*Cardiac Disturbances in Connection with Infectious Hepatitis. E. Adler and E. Lyon.—p. 111.

*High Oxygen Concentration Under Normal and Increased Respiratory Pressure in Cardiac Pain and in Pulmonary Edema. Inga Lindgren. —p. 127.

Unusual Electrocardiographic Pattern of Myocardial Infarction (Type QT_1C_{3-6}). V. Puddu, A. Mussafia and G. Giordano.—p. 133.

2-Thiouracil in Heart Failure and in Angina Pectoris. A. Sanabria. —p. 143.

Cardiac Disturbances in Infectious Hepatitis.—Adler and Lyon report 8 cases in which infectious hepatitis and jaundice was accompanied with electrocardiographic and subjective signs of myocardial lesions. The author directs particular attention to prolongation of the QT time and elevation of the T wave.

High Oxygen Concentration in Cardiac Pain and Pulmonary Edema.—Lindgren studied the effects of 100 per cent oxygen under normal and increasing respiratory pressure in patients with cardiac pain and in pulmonary edema. Pain in angina pectoris and in myocardial infarction was relieved by 100 per cent oxygen, and the electrocardiogram improved in angina pectoris. Electrocardiograms with the typical signs of coronary insufficiency during rest showed improvement during such oxygen administration. One hundred per cent oxygen at an increased pressure of about 10 mm. of mercury has been used with good results in cardiac failure with acute pulmonary edema. The treatment has been life saving in eclampsia complicated with pulmonary edema. In pulmonary edema the increased pressure is of value by increasing the oxygen content of the blood and by diminishing the venous return.

Nordisk Medicin, Gothenburg

32:2797-2836 (Dec. 6) 1946. **Partial Index**

Experiences and Impressions in Ophthalmologic Studies in U. S. A. in Spring of 1946. Gunnar von Bahr.—p. 2797.

Hospitalstidende

Determination of Meulengracht's Jaundice Index by Aid of Pulfrich's Stufenphotometer. P. H. D. Waagstein.—p. 2805.

Methylthiouracil in Continuous Treatment of Exophthalmic Goiter. E. Meulengracht, K. Kjerulf-Jensen and K. Schmidt.—p. 2806.

*Some Experiences with Propylthiouracil in Treatment of Exophthalmic Goiter. K. Kjerulf-Jensen and E. Meulengracht.—p. 2809.

Effect of Thiouracil Combinations on Fetus. E. Freiesleben and K. Kjerulf-Jensen.—p. 2811.

Norsk Magasin for Lægevidenskapen

Is Mantoux Reaction Reliable as Sign of Resistance to Tuberculous Infection? A. H. Brinchmann.—p. 2815.

Hygiea

Case of Celiac Disease in Uniovular Twins. S. Labdensuu and O. Somersalo.—p. 2819.

Propylthiouracil in Treatment of Hyperthyroidism.—Kjerulf-Jensen and Meulengracht's preliminary evaluation of propylthiouracil compared with methylthiouracil is based on the results of treatment of 32 patients with hyperthyroidism. They increased the original dosage to 0.1 Gm. three times daily, or 40 per cent of their usual methylthiouracil dosage. Even the most resistant case reacted to the treatment, but in 2 instances the thyrotoxic sympoms failed to decrease notably in spite of a month's treatment; further treatment of these patients with methylthiouracil gave good results. There was no indication that propylthiouracil possesses any fundamental effect different from the known effect of methylthiouracil. Partly because of the smaller amounts used, there were fewer by-effects after propylthiouracil than after methylthiouracil. Typical drug fever was seen in only 1 patient, who was also sensitive to methylthiouracil.

32:2837-2900 (Dec. 13) 1946. **Partial Index**

Medical Studies in the United States. J. Waldenström.—p. 2837.

Hospitalstidende

Albers-Schönberg's Marble Bones (Osteosclerosis Fragilis Generalisata): Two Personal Cases, One Treated by Neurosurgery. I. Søndergaard. —p. 2843.

Localization of Antianemic Principle in Human Stomach: I. Antianemic Effect of Powdered Human Fundus. E. Landboe-Christensen.—p. 2846.

Hygiea

*Pathophysiology and Therapy in Adynamic Ileus, Postoperative and in Peritonitis. Yngve Edlund.—p. 2849.

Svenska Läkaresällskapets Förhandlingar

Some Proposals for Reforming Medical Training. H. Malmros.—p. 2877.

Pathophysiology and Therapy of Adynamic Ileus.—Edlund says that circulatory changes in the intestinal blood vessels due to reflex responses to stimulation of the peritoneum are most important in the pathogenesis of adynamic ileus. They cause decreased intestinal motility, disturbed resorption of the stomach and intestinal contents, changes in the composition of the blood with release of water, electrolytes and plasma albumin from the blood through the capillaries, and, as a result of the diminished blood volume, a state of shock. In the first phase of treatment circulatory disturbances are counteracted by supplying plasma, by mechanical drainage of the gastrointestinal canal and by oxygen inhalation. In addition, salt and glucose are given parenterally to counteract dehydration and chloropenia. In the second phase of treatment amino acids and glucose are administered to prevent inanition and proteinemia; later a diet rich in calories and especially in proteins. Careful preoperative treatment and choice of anesthesia, together with adequate postoperative treatment, are important in preventing postoperative adynamic ileus. Toxemia is an added factor in adynamic ileus of peritonitis. Intensified administration of carbohydrates and protein is indicated to increase the patient's general resistance and the resistance of the liver and to accelerate detoxification. Further, therapy with sulfonamide preparations and penicillin may be considered.

Book Notices

Western Reserve University Centennial History of the School of Medicine. By Frederick Clayton Waite. Cloth. Price, $6. Pp. 588, with illustrations. Western Reserve University Press, 2073 Adelbert Road, Cleveland 6, 1946.

This history of a medical school is not simply a parochial account of local events of interest only to the old grads but is also stimulating to the general medical reader. The volume is much more than the history of a medical school, as might well be anticipated from the scholarly pen of Frederick Clayton Waite, who occupies an esteemed position as a medical historian. The hundred years of this medical school, since its origin in 1843, is pictured in the setting of medical education in the country at large. Western Reserve shared the vicissitudes and irregular progress of other medical schools. An example of historian Waite's eagerness to relate his school to significant and often previously unrecorded events in the history of medical education is seen in the opening sentence of his chapter on homeopathy: "This chapter is inserted in a volume otherwise concerned with regular medical colleges because no printed account exists that covers the entire period of homeopathic medical education in Cleveland." The author is rightfully proud of the history of Western Reserve University School of Medicine, quoting Flexner as saying to the university president, after the famous survey, "The Medical Department of Western Reserve University is, next to Johns Hopkins University, which for various reasons occupies an exceptional position, the best in the country." Yet the pride of local accomplishment emerges from the book as merely a part of the author's appreciation of advances in medical education everywhere. The broadness of his approach will appeal to any one interested in the history of medical education.

Urgent Surgery. Volume I. Edited by Julius L. Spivack, M.D., LL.D., Associated Professor of Surgery, University of Illinois College of Medicine, Chicago. Cloth. Price, $10.50. Pp. 714, with 572 illustrations. Charles C Thomas, 301-327 East Lawrence Ave., Springfield, Illinois, 1946.

This is a comprehesive book covering a large field in surgery. Urgent surgery is defined as that type of surgery which is not traumatic or elective but comprises cases in which operation becomes imperative within a matter of days. The first four chapters cover blood transfusion, anesthesia, all types of intravenous infusions, shock, hemorrhage, septicemia and gas gangrene. These subjects are presented as one would deal with them in urgent surgery as defined by the author. The chapter on laparotomy is one of the best. It explains what is meant by laparotomy and stresses the important fact that if such a procedure is undertaken the operating surgeon must be equipped and qualified to deal with major pathologic conditions. It also covers the various types of incisions and their closure and takes up the question of drainage. Three chapters are then devoted to surgery of the stomach and duodenum, including gastrotomy and pyloric stenosis. The subject is handled in routine fashion, similar to most textbooks on surgery. In dealing with gastrotomy the Spivak tubovalvular gastrotomy is stressed, explained and illustrated in full detail.

The chapter dealing with the pancreas is well written and covers the field in a concise and clear manner. Carcinoma of the pancreas and adenomas of the pancreas are mentioned. As these lesions do not come under the category of urgent surgery the treatment is merely mentioned without going into detail.

A very full, long chapter is allotted to urgent surgery of the liver and bile ducts, covering the field thoroughly. The author strongly recommends the costal incision for most biliary surgery. Several controversial subjects are brought up. One is that the author prefers operating on the gallbladder before opening the common duct. His reasons are that he gets better exposure of the common duct and a wider operative field and that he removes a potentially infected organ before operating on the common duct. He stresses the importance of electrocoagulation of the gallbladder wall adjacent to the liver. He terms this "mucoclasia." In this chapter he briefly mentions thrombosis of the veins of the lower extremities as a possible complication and advises the rather unusual treatment of elevating the legs on a splint and using ichthammol ointment dressings.

The field of appendicitis is covered completely. Two controversial subjects are handled: how to manage the appendical stump (he advises the true inversion without suturing) and drainage. He advocates the use of drainage routinely in appendical abscesses and in those cases in which satisfactory peritonization cannot be accomplished.

The chapter on intestinal obstruction covers the field thoroughly. When resections are indicated the closed type of anastomosis is favored. The last few chapters deal with urgent surgery in infants and surgery of the colon. The last two chapters cover perforated peritonitis and nonperforated peritonitis. From the author's remarks in several places in the book, especially in the chapters on laparotomy and appendicitis, he seems to favor early ambulation of operative cases.

The illustrations are plentiful and well done. They are used mainly to illustrate various surgical technics.

The book covers the field of urgent surgery in a detailed and competent manner, including preoperative diagnosis and preparation; also the surgical technic involved in the various operations. It follows through with postoperative care. This book is ideally suited to the use of residents in surgery. It is too far advanced for medical students. For the experienced operator it will be of interest, but it does not cover any one subject profoundly enough to be used as a book of reference.

Daniel Coit Gilman, Creator of the American Type of University. By Abraham Flexner. Cloth. Price, $2. Pp. 173, with 3 illustrations. Harcourt, Brace and Company, 383 Madison Ave., New York 17, 1946.

The life and the scholarly and academic ideals and accomplishments of Daniel Coit Gilman, first president of Johns Hopkins University, are described in this stimulating volume. When Gilman accepted this post in 1875 there was no true university in his sense, or in the European sense, in this country. Gilman's ideal was the establishment of an institution in which scholars "should be free to pursue their own researches, stimulating their students to prosecute study with a truly scientific spirit and aim." Gilman was not interested in just another undergraduate college whose professors would be burdened with "doing the duty of schoolmasters." His ideas were immediately accepted by the board of trustees and were financed with the (then) munificent endowment of $3,500,000, most of which was in Baltimore and Ohio stock, which some years later became worthless. The spirit of Gilman carried this idea into full fruition in the quarter century of his sojourn at Hopkins and set a pattern for graduate education in this country, emulated since in our better universities and research institutes. Gilman's emphasis was on the securing of promising men and providing them with opportunities for research productivity. Nowhere in the university was this principle more effectively applied than in the medical school, where Gilman added such scholarly names as Welch, Osler, Halsted, Kelly, Mall, Howell and Abel. His achievement in the medical school was especially notable, since medical education in America at that time was a sorry spectacle. The story is well told by one who was first inspired by Gilman's ideals and who himself became a master. It will interest every one concerned with higher education and medical education.

The Anatomy of the Bronchial Tree with Special Reference to the Surgery of Lung Abscess. By R. C. Brock, M.S., F.R.C.S., Surgeon to Guy's Hospital, London. Fabrikoid. Price, $12. Pp. 96, with 142 illustrations. Oxford University Press, 114 Fifth Ave., New York 11; Amen House, Warwick Sq., London, E. C. 4, 1946.

The anatomy of the bronchial tree has received attention by various authors. But this volume describes the unique anatomy of the bronchial tree. The author has made dissections of all the bronchi and has made metal casts of them. In addition he has made detailed bronchographic studies in the living subject to verify the interpretations which he presents in this volume. The illustrations are beautifully executed from the standpoint of photography and of artistic value. Many colored plates clarify the description in the text. The cost of producing these numerous plates, drawings and x-ray films must have been considerable. The Clinical Research Committee of Guy's Hospital

gave a generous grant to the author in order for him to be able to present this beautiful work.

While the subject of the book is the anatomy of the bronchial tree, the purpose of the author appears to be the relationship to the causes of abscess of the lung and the methods of exact localization. With this in mind the work demonstrates most beautifully the anatomy of the bronchi. In addition to the approach to the localization there are numerous drawings from bronchoscopic appearances of the various larger bronchi. It is interesting to note that in the description of the lymph nodes at the hilus of the lung the author uses the term "lymph glands." This term is obsolete in this country.

The photographs of the x-ray films are in the positive. In the American literature one almost always finds the reverse, the negative film being demonstrated. It is interesting to note that the author rarely uses stereoroentgenograms but prefers to use many positions and particularly the lateral view together with the anteroposterior position. The author is convinced that abscesses of the lung are produced by direct aspiration into the various bronchi and states that the upper lobes of the lung are involved almost as frequently as the lower, depending on the position in which the patient may be lying following operations.

With regard to embolism as a cause of abscesses, the author states that "this misconception is held by many people, and the object of this chapter is to show that the causal factors responsible for the development of an abscess in any particular site in the lungs are chiefly inhalation and posture, and that far from disproving the hypothesis of bronchial embolism the frequent occurrence of abscess in the upper lobes is the greatest single piece of evidence in favor of bronchial embolism and against vascular embolism."

The author quotes the Rabin method of localizing abscesses frequently and much of the presented material shows the value of the injection of iodized oil and methylene blue at the area where the abscess is outlined previous to operation. The pagination is unique, the pages of the reading matter being numbered independently of the illustrations. This book is recommended highly to all physicians who are interested in diseases of the lungs, particularly thoracic surgeons, physicians and roentgenologists.

The Eye Manifestations of Internal Diseases. By I. S. Tassman, M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania, Philadelphia. Second edition. Cloth. Price, $10. Pp. 614, with 243 illustrations. C. V. Mosby Co., 3207 Washington Blvd., St. Louis 3, 1946.

This edition is somewhat enlarged by additions of material on epidemic keratoconjunctivitis, toxoplasmic encephalomyelitis and other subjects. The author states in the preface that he has added descriptions of ocular allergy. He has included only tuberculous uveitis, conjunctivitis and keratitis but does not mention others which are more important in "bridging any existing gap between the eye manifestation and other aspects of internal disease." There are numerous omissions which should be included in such a work. Under metastatic gonorrheal infection there is no mention of dacroadenitis. In the section on macular degeneration, more lengthy discussion should be made of heredomacular disease, senile macular degeneration, familial macular degeneration, Noguchi's disease, central angiospastic retinitis and other conditions. A full discussion of the relation of the eye to headaches and dizziness should have an important place in such a book. On page 167 under treatment of cataract the author states that regular examination should be more frequent as the cataract approaches maturity. The stage where close observation is necessary is during the much earlier intumescent stage when there is danger of glaucoma. On page 165 is the statement that "complicated cataracts are also considered as secondary cataracts." Secondary cataract is a term which should be used only for the membrane, capsule and organized cortex remaining after an extracapsular lens extraction or injury to the lens. On page 194 about angioid streaks it is stated that it is associated with diabetes, Paget's disease and inflammations of the choroid. No mention is made of pseudoxanthoma elasticum, which is much more commonly seen with angioid streaks than all the others combined. The book will have a wide popularity because, for the time being, there is no better one on the market.

Science and Scientists in the Netherlands Indies. Edited by Pieter Honig, Ph.D., and Frans Verdoorn, Ph.D. Natuurwetenschappelijk tijdschrift voor Nederlandsch Indië, Volume 102—Special Supplement. Cloth. Price, $4; £1.1.0. Pp. 491, with 134 illustrations. Board for the Netherlands Indies, Surinam and Curaçao, 10 Rockefeller Plaza, New York 20, 1945.

According to the editors this volume is intended to present a picture of the development and status of various branches of the natural sciences, pure and applied, in the Netherlands Indies. They call attention to the fact that the book was prepared during the military occupation of both the Indies and the Netherlands, which made it impossible to obtain the collaboration necessary for a complete history of science in the Netherlands Indies. The book is made up of original articles and reprints of previous publications dealing with the development or status of various branches of science in the Netherlands Indies and also of travel accounts, biographic sketches, reviews and a list of scientific institutions, societies and workers at the time of the Japanese invasion. The editors are to be congratulated, for they have produced a valuable book which should fulfil their hope that this volume might be of use and interest to visiting civilians and members of the armed forces of North America, Great Britain and other countries. Moreover, it affords a valuable reference work which should be useful to scientists working in many fields throughout the world, especially to those who are concerned with the future progress of international health and human welfare.

Squint and Convergence: A Study in Di-Ophthalmology. By N. A. Stutterheim, M.D. Cloth. Price, 15s. Pp. 95, with 41 illustrations. H. K. Lewis & Co., Ltd., 136 Gower St., London, W. C. 1, 1946.

This small volume is a culmination of work done and articles printed in the literature over a period of the last twenty-five years. The trend of the author's thoughts and somewhat radical ideas were presented in a previous volume entitled "Indications for the Kinetic Treatment of the Eyes." The basic idea of this new physiologic approach to the correction of convergent strabismus is that fusion is a function of the nervous system integration and therefore is wholly independent of any refractive error. A battery of prisms is passed before each eye in increasing strength until the patient's abduction or divergence insufficiency is overcome. The refractive error remains uncorrected. The last half of the volume is completed with case reports and results. Stutterheim's colleagues have shown little desire to follow his work, either to corroborate it or to find fault. Until others have taken the time and effort to verify his findings, the work must be looked on as a most interesting experiment.

The Thyroid Gland in Medical History. By Alfred H. Iason, M.D., Instructor of Anatomy, New York Medical College, New York, Fabrikold. Price, $3. Pp. 136, with 50 illustrations. Froben Press, 4 St. Luke's Place, New York 14, 1946.

Iason has compiled a book that will be found useful to the specialist in thyroid physiology or pathology, for it apparently provides all the basic information about the development of our knowledge of the thyroid gland from the Egyptians to our day. The book is well illustrated, the paper is excellent and the format such that it can be read with comfort. The author has put his material together in a most orderly fashion, but in so doing the book, from the point of view of the nonspecialist reader, loses background. Focusing wholly on the thyroid, one has the impression that other actors too must have been concerned in the play. But to trace them would have made a much bulkier book and might have diverted the reader from the subject of the dissertation. As it is, the volume is a worth while contribution to our knowledge.

Studies of Compulsive Drinkers. Part I. Case Histories. By Herman Wortis, M.D., and Leonard R. Silliman, M.D. **Part II. Psychological Test Results.** By Florence Halpern, M.A. Edited by Jane F. Cushman, M.A., and Carney Landis, Ph.D. Foreword by Nolan D. C. Lewis, M.D. [Distributed by Quarterly Journal of Studies on Alcohol.] Paper. Price, $1. Pp. 90. Hillhouse Press, 432 Temple St., New Haven, Connecticut, 1946.

As usual, Florence Halpern's psychologic work and contribution are valuable in this monograph, containing in part I 18 detailed and interestingly presented case histories with clinical and test findings and in part II psychologic test results.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

HISTIDINE TREATMENT OF VASCULAR DISEASE

To the Editor:—**The treatment of vascular disease by the use of histidine and ascorbic acid was discussed in a recent article in The Journal (Wirtschafter and Widmann, March 1, 1947). The article did not describe the routine to be followed or the dose. Information on this subject would be appreciated.** Henry B. Gotten, M.D., Memphis, Tenn.

ANSWER.—The two compounds are administered in the following manner: All patients are started with an intravenous injection of 500 mg. of sodium ascorbate. This injection is a preliminary one and is not repeated thereafter. It is meant purely as an attempt to saturate the patient with ascorbic acid prior to the institution of the actual therapy. Immediately after the intravenous injection the patients are put on a regimen consisting of the intramuscular injection of 5 cc. of a 4 per cent aqueous solution of l(—) histidine monohydrochloride and the subcutaneous injection of 100 mg. of sodium ascorbate. These injections must be given separately. The gluteal muscle for the injection of the histidine compound and the area of the deltoid for the injection of the sodium salt of ascorbic acid are preferred. The reason for giving these injections separately is to prevent the reaction from proceeding at too rapid a rate. Though in the original article the time intervals between the administration of the two compounds varied from every four to every twelve hours, it is now believed that it is best to give the drugs every four hours day and night. If this is not possible, the drugs should be given not less often than every six hours day and night. Any longer time intervals will only prolong the time of healing and sometimes triple the patient-hospital days. Along with the injections, all patients receive 200 mg. of ascorbic acid by mouth, three times daily, a total of 600 mg. orally. When open lesions exist, either wet dressings in the form of boric acid or packs of isotonic solution of sodium chloride are used continuously or the wound is dressed with petrolatum gauze daily. Depending on the nature and severity of the wound, wet dressings may have to be repeated two to four times during a twenty-four hour period. The sodium salt of ascorbic acid must be used. The methyl glucamine salt of ascorbic acid and the sodium bisulfite salt of ascorbic acid have been tried and both of these have been found to produce a definitely lessened reaction. This has been confirmed by the patients themselves. The solutions used must be clear. Ascorbic acid solution which is definitely yellow is not as active as the clear solution. Similarly, it must be emphasized that the histidine solution should be clear and not a light brown, nor should it contain a brownish precipitate.

DANGERS OF PINWORM FISH FOOD

To the Editor:—**Pinworms are commonly cultured as food for tropical fish. Is this type of pinworm capable of infesting man? Is the use of this pinworm for fish food hazardous?** M.D., Michigan.

ANSWER.—Dealers advise that the "pinworms" cultured as food for tropical fish belong to the genus Tubifex and related genera. These worms are free living annelids and are not related to the pinworm which infects man. This worm, Enterobius vermicularis, is a strictly parasitic form and has been reported only from man and the higher apes. Effort has been made to infect experimentally several other animals without success. Under the circumstances there is no hazard in the use of the free living annelids mentioned as fish food.

POSTOPERATIVE IRRADIATION OF CHOROIDAL MELANOMA

To the Editor:—**What is the present status of postoperative irradiation by radium or x-rays of melanoma of the choroid?** M.D., Pennsylvania.

ANSWER.—Good evidence that postoperative irradiation is of value in the treatment of melanoma of the choroid does not exist. However, its use must be considered with the view that it may possibly be effective.

HONEY FOR TREATMENT OF PEPTIC ULCER

To the Editor:—**A man with a peptic ulcer has been under rigid management for many years, with only fair success. Seven months ago he began taking honey as a therapeutic measure. He is now on a normal diet and has not had symptoms since starting honey. Two friends of his have also started this diet of honey and whatever else they want to eat. They report that they are also symptom free. What is the therapeutic rationale of using honey in the treatment and management of peptic ulcer?** M.D., Wisconsin.

ANSWER.—The natural course of chronic benign peptic ulcer is characterized by periodicity of symptoms for reasons not clearly understood. This together with the important although intangible effect of mental suggestion renders the evaluation of any treatment difficult. (Palmer, W. L., in Portis, S. A.: Diseases of the Digestive System, ed. 2, Philadelphia, Lea & Febiger, 1944, p. 219). The patient and his 2 friends are convinced, no doubt, that honey is an "ulcer cure." The possibility that all 3 had spontaneous remissions would seem an unsatisfactory explanation. On the other hand, honey has a variable composition. The chief constituents, dextrose and levulose, do not have any specific therapeutic effect in peptic ulcer. To attribute the relief of ulcer symptoms to the use of honey per se would also seem an inadequate explanation. In this connection it is interesting to note that of a group of 20 ulcer patients treated with daily injections of 5 cc. of distilled water 12, or 60 per cent, became symptom free. Five, or 25 per cent, became symptom free after the first injection (Sandweiss, D. J.: Treatment of Gastroduodenal Ulcer with Histidine Monohydrochloride, THE JOURNAL, April 25, 1936, p. 1452). Such observations emphasize the difficulties in evaluating ulcer treatment. They also illustrate the importance of mental suggestion, enthusiasm, faith or whatever term one wishes to apply to the frequently observed phenomenon of "ulcer cures" following nonspecific therapy. The importance of these intangible factors in the treatment of peptic ulcer is well recognized even if not clearly understood. Consequently the therapeutic rationale of the use of honey seems quite clear. One can hardly escape the conclusion that it is only of psychotherapeutic value.

DERMATITIS IN MEAT HANDLER

To the Editor:—**A woman worker in a lunch-packing factory develops a severe acute contact dermatitis involving the face and neck and arms (all exposed areas of the body) whenever she works at her usual job making hamburger "patties" from meat (which is "untreated" at the plant). The hands, including the dorsal surfaces, are entirely clear during the handling of the meat. Roach powders and fly sprays, which are used from time to time at the factory, have been eliminated as a cause. That the dermatitis is caused by something at work is proved by the fact that it disappears after two weeks at home and treatment with the usual wet compresses and recurs one hour after she returns to her job. A warm air blower which is the only spray in the plant is used to blow out the grinding machine. (No chemicals are used.) The patient does not have an allergic history and has eaten meats of all kinds all her life. Ordinary lighting facilities are used in the plant.** M.D., Ohio.

ANSWER.—There can be but little doubt that the patient described in the query has an occupational dermatitis, as shown by the fact that she gets well when she is away from work and the eruption returns when she goes back to work. In order to find the actual irritant in her occupational environment which causes the eruption it would be necessary to make an exhaustive study of the substances with which she comes in contact. "Patties" are not entirely excluded in spite of the fact that she has eaten meats of all kinds, because hamburger patties may contain spice and other substances which she does not ordinarily eat. Besides, it is possible to develop a dermatitis from contact with raw meat and yet not develop dermatitis when the same meat is cooked and eaten.

The warm air blower also should be investigated, because instances have occurred wherein warm air brought from one part of the plant has carried an irritant to another part of the plant. Cases have also occurred wherein warm air flowing in the direction of a cold area has been known to pick up irritants on the way, which have caused dermatitis on workers in the area of final distribution of the warm air. A series of patch tests as a result of such an investigation may reveal the actual irritant.

As preventive measures while working, it is suggested that impervious sleeves made of a plastic such as vinylite be worn on the arms, and that the face and neck be covered by a protective ointment such as equal parts of lanolin and cold cream and that before the lunch hour the ointments be washed off and reapplied immediately after lunch.

Reference:

Schwartz, Louis; Tulipan, Louis, and Peck, S. M.: Occupational Diseases of the Skin, Philadelphia, Lea & Febiger, 1947.

DIAGNOSTIC PROBLEM—POSSIBLE EPILEPSY

To the Editor:—A man aged 24, about 5 feet 6 inches (168 cm.) tall, weighing 175 pounds (79 Kg.), was involved recently in an airplane crackup. He was taken from the plane in shock, which was of eight hours' duration. He was given intravenous saline solution for three days and epinephrine and morphine injections. His previous history included nervousness, contusions and lumbar strain. The blood pressure is 100/68. Since this accident, whenever the patient is under tension and becomes nervous he begins shaking and "blacks out." He becomes cold to such a degree that his teeth chatter and he attempts to pull himself together in an effort to get warm. He has severe headaches and vomits. His knees give way under him. His entire body becomes red and even his eyes are bloodshot. It is my impression that the blood vessels become dilated. It seems as if one big hive was manifesting itself. If he tightens his face, clenches his fist and tautens his muscles or skin, the area becomes white. The patient appears to be in extreme shock during this period. In recovery there are pains in the knees and pins and needles sensations in the fingers. At cessation of the spells he feels well enough to participate in any form of work or play. Three of these spells occurred during the last week—one at a football game, one at the table and the third while making a hard shot at pool. Is this a petit mal epilepsy?

Morris H. Pett, M.D., Gloucester, Mass.

ANSWER.—Before a satisfactory diagnosis can be made further studies should be carried out including the blood pressure and blood sugar during and following an attack, x-ray examination of the skull, examination of the cerebrospinal fluid including that of the pressure, cells, protein, Wassermann and colloidal gold, and a complete physical and neurologic examination. It may also be desirable to perform a ventriculogram or an encephalogram.

The possibility of a "diencephalic epilepsy" such as was described by Wilder Penfield (*Arch. Neurol. & Psychiat.* **22:** 358 [Aug.] 1929) must be considered. Such attacks might arise from a tumor in the third ventricle. Such a lesion would also account for the associated severe headaches and vomiting—a paroxysmal hypertension from an adrenal tumor seems unlikely unless the phase of vasodilatation is secondary and is preceded by one of vasoconstriction. In many respects the attacks suggest the response to an intravenous injection of histamine, and this may be an unusual histamine reaction. The reason for such a condition is of course not obvious.

STABILITY OF MORPHINE AND ATROPINE SOLUTIONS

To the Editor:—How long will ampules of morphine sulfate or morphine sulfate and atropine preserved in chlorobutanol and sodium sulfite keep? Is it safe to use them at all after several years? How soon will their potency diminish?

Anthony M. Susinno, M.D., Palisades Park, N. J.

ANSWER.—Solutions of morphine sulfate are fairly stable and would probably keep indefinitely in ampules or if protected by chlorobutanol and sodium sulfite against bacterial or fungous contamination. A 1 per cent solution of morphine hydrochloride is official in the British Pharmacopeia. It contains 25 per cent of alcohol. Atropine sulfate solutions deteriorate quite rapidly. Sollmann's Manual of Pharmacology, sixth edition, page 376, quotes Simon, 1915, that there is considerable loss of activity even in three days, and Joachimglu, 1915, that sterilized solutions in ampules retain their full activity for at least four weeks. The rate of change varies with conditions which are not always predictable. The change involves only the potency; the toxicity would not be increased. It would be quite safe to use the ampules after several years, but they may not be fully active.

ELECTROCOAGULATION FOR POSTTONSILLECTOMY HEMORRHAGE

To the Editor:—Is electrocoagulation a satisfactory means for hemostasis after tonsillectomy? Is secondary hemorrhage more likely after electrocoagulation than after spontaneous clotting, sponge compression, hemostat crushing or tying? Can the concentration of ether vapor in the patient's alveolar air be such as to make the use of electrocoagulation in the mouth hazardous if, after the induction of deep surgical ether anesthesia, all anesthesia equipment is rpmoved from the operating room?

M.D., Texas.

ANSWER.—Electrocoagulation can be used to stop bleeding after tonsillectomy. It is not as satisfactory as sponge pressure, the hemostat or the ligature. The latter are simple, are easy to use and do not require special precautions as to anesthesia.

The methods used to obtain hemostasis vary with the experience and inclination of the operator. In general, one should attempt to obtain a dry tonsillar fossa, using methods that give minimum tissue injury and the shortest amount of time for anesthesia. Sponge pressure harms the tissues the least but is more time consuming and less sure. The hemostat is more accurate because it can be applied directly to the offending spot but, because of its crushing effect, there must be a certain amount of secondary tissue necrosis and sloughing and danger of bleeding later. The ligature that is tied over the end of a hemostat may come off; the ligature that is placed with a needle requires skill; the needle may break, and there is danger of introducing infection into the deep tissues of the neck.

All three methods may be employed, from the simple to the more difficult, as conditions demand. At all times as little trauma as possible is inflicted and, if ether is used, the time of anesthesia is made as short as possible.

If electrocoagulation is used for hemostasis, the least amount of current necessary is applied, so that tissue destruction is kept to a minimum. It can be a useful instrument in practice, although there are limitations. The apparatus often requires adjustment, and this means that one other person must be present besides the operator. In an adult, or when a local anesthetic has been used, there may be little difficulty in gaining access to the desired area. However, a child with a metal mouth gag in a small mouth offers difficulties in approach and opportunities for short-circuiting and burning of the lips and gums. Before using electrocoagulation one should try simpler and less dangerous methods.

Secondary bleeding follows electrocoagulation more often than after the use of other methods; and, in addition, this bleeding is apt to be more stubborn.

The hazard of explosion is definitely present if electrocoagulation is attempted in the course of an ether anesthetic unless the patient is allowed to waken to such an extent that the ether concentration in the lungs is minimal. At this stage the patient is not anesthetized to a worth while degree, from a pain killing point of view. Also at this stage he is apt to retch, vomit and be so active and uncooperative as to make a painstaking procedure difficult, if not impossible.

BRIDGING OF THE SELLA TURCICA

To the Editor:—A man has had chronic headaches for years. Roentgenograms reveal calcification of the sella turcica or rather a bridging over of the sella. What is the significance of this finding and can it be the cause of his headaches? What is the treatment?

Arthur Henrichsen, M.D., Van Nuys, Calif.

ANSWER.—Bridging of the sella due to close approximation of the interior and posterior clinoid processes is found normally and usually is not significant. However calcification in, above, below or to the side of the sella turcica is very significant. It is found in (1) a calcified internal carotid artery, (2) suprasellar cysts, (3) intrasellar tumors such as adenoma or glioma, (4) meningioma and (5) glioma of the optic chiasm. The patient described in the query should have repeated testing of his visual fields, i. e. every 15 degrees, for form and color. Both optic nerve heads should be examined for optic atrophy or papilledema. Glucose and insulin tolerance tests also should be made. If all these tests are within normal limits then other causes should be sought, such as hypertension or arteriosclerosis.

VISION TESTS

To the Editor:—What are the advantages of the Telebinocular (Keystone Company) or the Massachusetts vision test (Welch Allyn Company) over the Snellen Chart for testing school children?

D. H. Lawler, M.D., Hammond, Ind.

ANSWER.—In the visual examination of school children the advantages of the Telebinocular, which is basically a stereoscope, are that (1) little space is required for use, (2) it combines reasonably accurate tests for distance visual acuity, muscle balance, color vision and stereopsis and (3) it facilitates rapidity of examination, especially in groups of 15 or more, and in older children and adults. Among its disadvantages are (1) expense, (2) limited adaptability for small groups and (3) possibility of misinterpretation of the information afforded.

The advantages of the Massachusetts vision test are that (1) it is designed especially for the examination of school children and is well adapted for those in the primary grades, (2) it combines an accurate test for visual acuity and muscle balance both for distance and near and affords a possible separation of hypermetropes from myopes and (3) it facilitates rapidity of examination especially with large groups. Among its disadvantages are its cost and the advisability of having trained help in its use.

The Snellen Chart has the advantages of being easily procurable, inexpensive and easy to install. Small groups may be practically tested for distance visual acuity with literate or illiterate Snellen Charts if intelligently used. Among its disadvantages are that (1) variations of illumination are often associated with its use, (2) its letters and figures are easily memorized and (3) its usefulness is limited to distance visual acuity.